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Original Communications

PREGNANCY AND HEART DISEASE FROM A MEDICAL VIEWPOINT*

REPORT OF A STUDY OF FORTY CASES FROM THE SLOANE MATERNITY HOSPITAL, NEW YORK CITY

BY W. W. HERRICK, M.D., NEW YORK, N. Y.

INTRODUCTORY

THE obstetrician of the period of medical development now passing, in addition to dealing with the mechanical problems of labor, has too often borne alone the burden of manifold and often vital decisions respecting the care of the infant and the numerous problems of pregnancy proper to internal medicine. Among these the acute infections, the toxemias, and the myocardial insufficiencies in relation to pregnancy, parturition, and the puerperium are of such complexity and importance as to call forth the best endeavor of those practiced in both fields. In recognition of this fact, in the Sloane Hospital for Women during the past two years, a combined study of these problems has been made by the obstetrician and the internist. In publishing the result of these studies, the last named—the myocardial insufficiencies—will be first considered. The acute infections and the toxemias will be taken up in later papers. This paper, therefore, summarizes the experience with all cases showing symptoms of myocardial insufficiency delivered in the Sloane Hospital during the two years from October, 1919, to October, 1921.

*Read (by invitation) at a meeting of the New York Obstetrical Society, February 14, 1922.

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REVIEW OF LITERATURE

In reviewing the recorded experience of others in similar cases, one is impressed with the wide difference in results and opinions. A few examples may be quoted:

Harrar[1], in a report of seventy-five cases with broken compensation during pregnancy, found an immediate maternal mortality of 30 per cent. He expressed the opinion that the prognosis improves the further along in pregnancy the woman is at the time of the first breakdown in circulatory balance. In nine cases in which the breakdown was between the ninth and tenth month, there was one death; of nine with the breakdown before the sixth month, seven died. In women giving history of broken compensation in previous pregnancies or before the present one, the mortality increases 60 to 70 per cent. Age and parity have little effect except in primipara. Hearts with a double mitral lesion are more prone to fail and to recover. Simple aortic lesions are most infrequent and have best recuperative power. Combined mitral and aortic disease is the most serious of all.

Longaker[2] states that the endeavor to carry these patients over months of increasing circulatory burdens to the period of fetal viability by rest and cardiac tonics after serious symptoms have developed is prone to spell disaster. "In the face of decompensation, there remains but one thing—empty the uterus."

Manges[3] proffers the opinion that cases of mitral stenosis when accompanied by mitral regurgitation usually have no trouble in labor and that mitral stenosis of the uncomplicated type is the only lesion giving trouble in pregnancy.

Hussey[4] believes that the majority of women with compensated heart disease go through pregnancy and labor without signs of decompensation. He states that the majority of fatalities occur some time after labor and believes that pregnancy may draw appreciably upon the store of cardiac reserve. He considers that the uterus should be emptied if broken compensation occurs early in pregnancy; if the patient has had broken compensation in former pregnancies; or if the symptoms of decompensation persist despite treatment.

Citing numerous writers, Hirschfelder[5] gives a mortality of 3 to 61 per cent in cases of valvular disease of the heart as a complication of pregnancy. Newell[6] believes that valvular disease of the heart complicating pregnancy may, for therapeutic purposes, be divided into two classes: The first, those showing decompensation early in pregnancy. These should have early abortion. The second, those showing decompensation in the latter period of pregnancy. These should have a second stage shortened and made as effortless as possible. In a later article, Newell[7] states that three per cent of pregnant women have cardiac complications which may be expected to react more or less seriously to the strain of pregnancy and labor. This writer is impressed with the reduction in reserve power in the heart resulting from childbearing and states his belief that every cardiac patient must pay for her child some price in length of days.

Blacker[8], in a very satisfactory review of the entire subject, remarks: "It is certain that the majority of cases of valvular disease of the heart complicating pregnancy do perfectly well and pass through labor and the puerperium with no symptoms." In 453 collected cases, Blacker finds the mortality 12 per cent. He remarks that the nature of the valvular lesion is not of so much importance as the capacity of the myocardium—an opinion with which the majority of earlier writers are at variance—mitral stenosis being as a rule regarded as the more serious menace to the pregnant woman.

Feis[9] remarks upon the rarity of heart failure in pregnancy. He quotes the experience of Wissner in the Berne Frauenklinik, who in 4,000 births, noted but 25 such instances with but one fatality. From the reports of several observers, the same writer cites the following mortality rates in the condition under consideration: Wissner, 37.6 per cent; Schlayer, 48 per cent; Macdonald, 60 per cent; Leyden, 65 per cent; Guerard, 40 per cent; Schneider, 14 cases with one death; Benny Hart, 8 cases of mitral stenosis and 7 deaths; Vinay, in the Hotel Dieu, 20 cases and no deaths. In Schlayer's experience, but 46.5 per cent of children in cases of chronic valvular disease were carried to term. Twenty-nine per cent were born dead. Feis'[9] experience leads him to the dictum that the probability of the survival of the child is so dubious that its life should not weigh greatly in the treatment of the mother.

Recently, Mackenzie[10] has written at length on this subject. His conclusions are, in general, not at variance with those of other writers. Few of these writers have stressed the importance of the purely medical treatment of cardiac decompensation in pregnancy, excepting Mackenzie[11] who says, "By bitter experience general practitioners have recognized that in certain diseased states of the heart there is great danger to the life of the woman and child. For over 50 years there have been many attempts to find out where the danger lies, and to recognize the signs which foretell disaster and the signs which need cause no anxiety. Yet the most recent of our textbooks show no advance on the views of 50 years ago."

"There is a physiologic problem involved, but no physiologist has attempted its solution. There are problems intimately connected with clinical medicine, yet physicians have no opportunity for studying the pregnant woman. The obstetrician sees her, but no obstetrician has yet learned the elements of cardiac symptomatology sufficiently to enable him to acquire the necessary information. Here is a problem of the first importance, which will, time and again, confront every general practitioner, and in the whole hierarchy of a medical school there is not one teacher or even group of teachers, capable of acquiring, far less of imparting, the necessary knowledge."

Most of the case reports deal with the purely obstetrical side of the matter, in which delivery is paramount. The larger number of patients have entered the obstetric hospitals at or near term—often late in labor, and under circumstances not affording opportunity for adequate medical measures. It seems clear that, no matter how skillfully labor be guided, it is the tardy recognition and care which is largely responsible for the high mortality in the cardiac disorders of pregnancy. The advantage to the patient of careful oversight throughout the entire antenatal period with the prompt institution of medical treatment in infections, toxemias, and cardiac conditions is unquestionable. This is more than a matter of opinion; it is a fact capable of ample proof.

MANAGEMENT OF CARDIAC CASES IN THE SLOANE HOSPITAL

The general plan of management of the pregnant woman with chronic disease of the heart is as follows: Pregnancy being a physiological process, we have taken the position that in the case of chronic cardiac disease with broken compensation, it is not the pregnancy, but

the heart condition which is the primary source of trouble. Therefore, where possible, pregnancy has been temporarily ignored and the first endeavor is the restoration of cardiac compensation before considering delivery. Of the greatest importance in accomplishing a satisfactory end result is the early recognition of disorders of the heart in the antenatal clinic. Cases presenting heart lesions are observed at very frequent intervals, and at the onset of unusual dyspnea, edema, palpitation, or other signs of decompensation, are admitted to the hospital and treated as cardiac cases. We would emphasize the importance of not instituting any measure looking toward delivery of the patient while symptoms of decompensation are present. Our own experience and review of the experience of others, has convinced us of the danger in hastening delivery in untreated cases of cardiac decompensation and has furnished proof of the high mortality in cases of decompensated cardiac disease delivered promptly upon their admission to obstetric hospitals. The greatest safeguard of the pregnant woman with chronic cardiac disease is, therefore, the antenatal clinic and prompt hospitalization at the first signs of serious decompensation.

The details of the medical treatment of this series of cases may thus be outlined: Rest in bed is usually enforced, but if the patient is more comfortable sitting in a chair, this is allowed. We have felt that the absolute prohibition of all bodily effort is not in every case necessary, and that it is possible some minor activity is an advantage in promoting the peripheral circulation, favoring venous return and thus reducing the strain on the heart. Adjuncts of value are massage and passive movements.

The diet is an ordinary, simple, light, well-balanced ration, the fluid content of which is somewhat limited, the average allowance of fluid being about fifteen hundred cubic centimeters in twenty-four hours. In cases with edema and signs of circulatory stasis, the Karrel diet, consisting of one quart of milk in twenty-four hours without intake of other solid or fluid, may be given for from two to five successive days until results appear. If, on this diet, thirst is annoying, cracked ice, fruit pulp, or chewing gum may be allowed. It is our experience that the Karrel diet is as satisfactory in the anasarca of pregnancy as in that accompanying decompensation apart from pregnancy. That there is an increased blood volume in pregnancy seems well established and is undoubtedly of great importance, and its reduction is accompanied by a very great improvement in most cases. With this purpose in view, along with diminished fluid intake, restriction of salt is enforced. Hydrogogue cathartics are of use in a few cases as an accessory measure in this comparative dehydration.

Digitalis is the most valuable single agent in restoring compensation. In cases presenting only slight symptoms of decompensation,

digitalis is given by the so-called "small dose method"—fifteen to twenty minims of a standardized tincture being administered three or four times a day until physiological effects are obtained. In the more pronounced cases of cardiac insufficiency requiring more prompt effects, one dram of the tincture is given three or four times daily for one or two days, followed by smaller doses. In a very few cases presenting grave symptoms of cardiac decompensation, the Eggleston "body weight" method is employed. In this, fifteen cubic centimeters of a standardized tincture for each one hundred pounds of body weight are administered within a period of twenty-four hours. The initial dose is one-half the total quantity. The three later doses consist of one-half the remaining amounts given at six hour intervals. In only one case—an emergency—was strophanthin, one milligram, given intravenously. This was an example of acute cardiac dilatation accompanying influenza. It is probable that the method by which digitalis is given is, in most cases, unimportant. The aim is to administer sufficient to produce the desired physiological action.

When satisfactory digitalis results are secured, as shown by lowered pulse rate, lessened pulse deficit, improvement in subjective symptoms, and in the establishment of diuresis, the effect of the drug is continued by administration of the tincture, minims twenty, every twenty-four hours—an amount which the studies of Pardee[12] have shown adequate for the continued action of the drug in the individual of average weight when once digitalization has been accomplished. In this plan of treatment, diuretics play a small part but are often useful in initiating diuresis when digitalis effects have begun to appear. One or two doses of diuretin, grains ten, or theocin, grains ten, may be given with profit.

The response of the majority of pregnant women with cardiac decompensation to treatment of this kind, suitably modified to fit individual requirements, is most satisfactory and is further proof of the familiar statement that "the average case of chronic valvular disease bears pregnancy well." In general, in this treatment, pregnancy may be ignored. If proper response is had, pregnancy may be allowed to proceed, the patient generally returning to her home to report for examination at weekly intervals and to be again hospitalized with the return of decompensation. Frequently, a woman will be admitted several times for weekly periods during the later months of pregnancy for the restoration of circulatory equilibrium. By such a program of intermittent hospitalization, the majority of women with weak hearts can be carried along, decompensation kept in abeyance and term approached with the heart in condition to bear the strain of labor.

Not every case responds happily to medical treatment. When satisfactory results do not ensue, decision as to the proper procedure frequently taxes the wisdom of the most experienced obstetrician and

internist, and is a matter of judgment in the individual case. There are so many factors of weight in this decision that no formula applicable to all cases can be worked out. These factors may be brought under two heads: First, medical, including the efficiency of the myocardium as shown by the degree of decompensation, the response to treatment, and, in multipara, its behavior in previous pregnancies. Second, the obstetric, including the size of the pelvis, size of the fetus, stage of pregnancy, state of the cervix, parity, efficiency of the mechanism of parturition, etc. If decompensation is marked and persists despite careful medical treatment, pregnancy should be terminated by the method entailing the least effort on the part of the patient. Any other course is full of danger. Further, the high fetal mortality already mentioned (Feis[9]), argues against the continuation of pregnancy when decompensation is unyielding and severe. If decompensation occurs early in pregnancy and if former pregnancies have resulted in severe damage to the myocardium, abortion is generally advisable.

There are numerous cases in which cardiac symptoms do not become severe until the latter half of pregnancy and, while showing improvement, do not entirely disappear under medical treatment. A certain number of these borderline cases may be carried along with careful observation in the hospital to term or to fetal viability when normal labor or induction may be passed through with success. It may be said that more of these cases than seems possible go through successful childbearing without serious event. In making decision in cases of this sort, much depends upon the attitude of the woman. If, for the sake of offspring, she is willing to take the risk of acute dilatation during or after labor and of decline in cardiac efficiency afterward, this has a certain weight in the decision.

If the period of fetal viability is reached, further decision must be made. Shall the pregnancy proceed to term? If not, how shall it be ended? If obstetric conditions indicate the necessity of cesarean section and if decompensation impends, this measure may be taken so soon as a living child is assured. If, however, obstetric conditions promise a second stage free from undue effort and decompensation can be warded off, it is usually wiser to allow continuation to term when a more facile parturition is assured than to embark upon the uncertain course of an induced labor in advance of term. In certain cases at term in which an easy second stage is not probable, cesarean section may be the wise course even if the heart condition is well under control.

Upon the management of labor depends, to a large extent, the future efficiency of the myocardium. While the majority of women with heart disease will pass through labor with safety, a certain number will show permanent decline in reserve vigor of the heart. It is to

spare this that cesarean section is often resorted to. Again, this gives opportunity for sterilization when this seems advisable.

During labor, the physician is chiefly concerned with the shortening and lightening of the second stage. It is probable that the uterine contractions do not in themselves greatly burden the circulation. Aside from the circulatory demands of increased metabolism and the psychic effect of the pain, such contractions of masses of involuntary muscle may be disregarded as important factors in any circulatory disturbances accompanying pregnancy. It is the play of the entire mechanism of the second or expulsive stage that is the chief danger to the compromised heart. The contraction of the voluntary muscles, the forced depression of the diaphragm, the holding of the breath during efforts to expel the fetus, is a strain as real as it is immeasurable in precise terms of work performed. The expulsion of the fetus with the consequent decrease in intraabdominal pressure is itself a source of embarrassment to the circulation. The intrathoracic pressure relations are altered greatly and suddenly. As Pouliot[13] has insisted, the lower position of the diaphragm immediately following parturition has an effect on the lungs not unlike that following the removal of a large pleural effusion, under which conditions one may rarely observe an acute edema of the lungs. This, in Pouliot's mind, explains the pulmonary edema occurring so frequently immediately postpartum. That this sudden decline in intrathoracic pressure is more of a menace in mitral stenosis than in other conditions is understandable when we consider the greatly increased tension in the pulmonary circulation in this valve lesion. It would appear that herein lies one of the important causes which justifies the view that mitral stenosis is attended by more risk in childbearing than is any other heart lesion. The necessity of making this second stage of labor as short and easy as possible has long been recognized by obstetricians. The means of accomplishing this must vary with each case and are important enough to demand separate discussion.

COMPLICATIONS

With the completion of labor, the menace which threatens the decompensated heart is by no means abolished. The obstetrician is well aware of the possibility of an acute pulmonary edema within a few hours, or even twelve or fourteen days following parturition. The cause of this dangerous symptom is a matter of debate. Three factors, operating singly or in combination, seem important. We have already mentioned the sudden diminution of pressure upon the lung resulting from the expulsion of the fetus which, particularly in mitral stenosis, with its greatly increased pressure in the pulmonary circulation, may act in the same manner in promoting pulmonary edema as the with-

drawal of a large pleural effusion. Similar train of cause and effect might rarely be observed in mitral insufficiency as well.

The mechanical theory of Welch may be invoked as a fairly satisfactory explanation of certain types of pulmonary edema,—particularly those following severe toxemia with prolonged arterial hypertension. It is probable that sudden failure of the left ventricle under the strain with continued normal and, therefore, disproportionately great activity on the part of the right ventricle may result in great pulmonary congestion and transudation. This type of circulatory imbalance often occurs several days after parturition and especially with the establishment of lactation. The metabolic demands of this function, the redistribution or increase in amount of body fluids may, each or all, be factors. There would seem little question of the important rôle of increased blood volume in this type of pulmonary edema of pregnancy. The frequent practice of flooding the body with enormous quantities of water when toxemia is present is one that requires great discrimination. When toxemia is accompanied by arterial hypertension or circulatory stasis, great increase in fluid intake may spell disaster. Toxic effects on the endothelium of pulmonary capillaries increasing permeability is doubtless a real but variable and imponderable factor. So is the toxic degeneration of the myocardium in the so-called mechanical type. Bronchospasm as a cause of the pulmonary edema of childbearing seems of little importance.

The immediate treatment of severe pulmonary edema calls for prompt bleeding. During labor, this may be accomplished by promoting loss of blood from the uterus. Otherwise, venesection is demanded. Since reduction in venous pressure follows only after the withdrawal of sixteen ounces or more of blood from the individual of average weight, at least that amount should be taken. Morphine, grains one quarter, hypodermically is essential. Entire quiet is enforced. In cases with arterial hypertension, nitroglycerine is valuable. One one-hundredth grain is given every five minutes until effect is had. The further treatment of such a case is that of the decompensated heart after labor.

TREATMENT OF THE DECOMPENSATED CASE AFTER LABOR

This includes bed rest, the Karrel diet in cases with edema, otherwise moderate restriction of fluids, digitalis to tolerance, sedatives as required, and diuretin at intervals if indicated. Massage and passive movements are begun promptly. Later, exercise is allowed provided the response is satisfactory. If a given effort does not result in an acceleration of the pulse of more than twenty beats per minute, and if, after two minutes, the rate return to within five or ten beats of the previous resting rate, the bounds of cardiac capacity have probably not been passed. The more complex and, theoretically, more exact

tests of the functional capacity of the myocardium are probably no more satisfactory than this simple exercise test. Governed by this test, the patient may gradually resume activity. In general, about one month of rest in bed or chair, followed by graduated activity under careful supervision is necessary. In cases exhibiting dilatation of the heart, the return of the apex impulse to its former site, a change to a more normal character, and the disappearance of the murmurs indicating a relative mitral insufficiency, are important preliminaries to activity and must, in selected examples, supplement the exercise test.

Digitalis should be continued in therapeutic doses during this convalescent period. The person of average weight will tolerate about twenty minims of the standard tincture daily. Some will profit by larger amounts. Others will exhibit extrasystoles, ventricular tachycardia, nausea, or other evidences of toxic action, and should have the drug at intervals rather than by a continuous method.

Many women tend to gain weight unduly during the months following childbirth. The danger of this to the cardiac patient is obvious. In controlling this matter, studies of the basal metabolism and chemical survey of the blood are desirable and may unmask metabolic or endocrine defects of importance. Where such refinements are not available, quantitative restriction of diet with disproportionate reduction in carbohydrate often gives practical results that are satisfactory. Judicious exercise and thyroid extract given with caution may supplement this diet.

Anesthesia.—It is our opinion that much of the apprehension of the possible ill effects of anesthesia in instances of chronic heart disease is not justified by practical experience. In our series, a judiciously given ether anesthesia was, without exception, well borne. In these cases, ether has been preferred to other anesthetics by the operating staff.

Mitral Stenosis.—While the not infrequent statement that this lesion is incompatible with a safe pregnancy is certainly not warranted by experience, mitral stenosis should be regarded more earnestly than other valvular defects. This is especially true of cases giving such evidence of great narrowing of the mitral ring as a lengthy murmur and thrill extending throughout diastole, constant rapidity of the heart rate, cyanosis, exertional dyspnea, and signs of stasis however slight. In such cases, pregnancy should be avoided or terminated. Curiously, the danger of embolism does not seem to be increased by pregnancy.

One case of mitral stenosis (hospital number 44733) illustrated very convincingly the mechanism of the Graham-Steell murmur of relative pulmonary insufficiency. Before labor the signs were those of a marked involvement of the mitral valve with stenosis and insufficiency and accentuation of the second sound at the pulmonic area. After delivery, a soft diastolic murmur appeared at the pulmonic area and

in the third and fourth spaces to the left of the sternum. With this, the second sound at the pulmonic area became very faint. In ten days the diastolic murmur vanished and the pulmonic sound was again loud and distinct.

Auricular Fibrillation.—In 1921, Thomas[14] remarked that he could find in all the literature no "mention of cardiac arrhythmia in pregnancy or any cases of auricular flutter or fibrillation accompanying that state." To Thomas' report of a seriously decompensated case with fibrillation that went through labor without great embarrassment of the heart, I add four cases showing fibrillation of the auricle. All of these underwent therapeutic abortion excepting one in whom the arrhythmia was transitory and immediately followed labor. In this case, within a few hours, a normal rhythm was restored and no cardiac symptoms have since been experienced.

Auricular fibrillation being most often a sequel of marked mitral stenosis would, in general, interdict pregnancy. However, those rare cases with adequately maintained circulation and some margin of cardiac reserve power might bear children with reasonable safety.

Auricular Flutter.—This occurred once as an incident immediately postpartum. It vanished within a few hours and no trace of heart trouble could be detected several months later.

THE PERMANENT EFFECTS OF PREGNANCY ON THE DISEASED HEART

Do pregnancy and labor permanently reduce the reserve power of the diseased heart? Only careful study of cases for several months or years postpartum can give a satisfactory answer to this question. In general, experience indicates that the scriptural principle applies: "To him that hath shall be given, and from him who hath not shall be taken away even that which he hath." The woman with little dilatation and fair reserve power in the heart muscle may, six months after parturition, reveal no decline in circulatory efficiency, while the more gravely afflicted show permanent injury. Even among this latter class, however, are startling exceptions. One may see with astonishment a woman with a narrow mitral ring and permanent auricular fibrillation, or one with recurring and severe decompensation, pass through more than one labor without apparent increase in cardiac discomfort. Satisfying explanations of some of these cases cannot be given. Of one matter, however, we may be certain; such good results depend very largely upon the care given before, during and after labor. Of greatest importance is the prevention of serious decompensation by the prompt institution of medical treatment with the earliest onset of its symptoms. If this is accomplished, if the labor is made free from undue stress by appropriate obstetric measures, and if resumption of activity postpartum be carefully adjusted to the

capacity of the heart, the patient may, as a rule, look forward to a restoration of cardiac reserve power at or near the former level.

For this, there seems good reason. Pregnancy is a physiological process. Cardiac decompensation accompanying it is the result of added burdens largely of a mechanical sort, such as increased blood volume, increased body bulk, increased metabolic demands, high position of the diaphragm with its effect on heart and lungs. In the absence of toxemia, such decompensation is not the result of any inflammatory or degenerative process involving the myocardium such as so generally underlies the cardiac failure of most other states. The therapeutic problem is, therefore, measurably simpler, more promising and more successful in pregnancy than under most circumstances.

Examination of some thirty of our cases from six weeks to twelve months postpartum reveals the satisfactory fact that there are few exceptions showing either by history or physical signs appreciable decline in circulatory efficiency as a result of the pregnancy. Exceptions have been those becoming obese, those suffering further inroads of rheumatic infection and one example of syphilitic aortitis that has continued on the expected downward course.

Details of the forty cases upon which this paper is based are found in the accompanying tabulation. Of the two deaths, one was a case of influenzal pneumonia with decompensated heart and edema of the lungs. She entered in a moribund state and died shortly after admittance. The second death was in a case of marked mitral stenosis delivered in the Sloane Hospital one year before. This patient was advised to consent to termination of pregnancy, but refused, dying from acute dilatation the day following admission.

THE EFFECT OF CHRONIC HEART DISEASE UPON THE FETUS

In this series of forty cases, there were ten fetal deaths. Of these, three were therapeutic abortions, four were stillbirths, one died about one hour after birth and one was a stillbirth following a postmortem cesarean section. One maternal death occurred without delivery. As might be expected, the fetal mortality was greatest in mitral stenosis with concomitant toxemia of pregnancy. The influence of chronic valvular disease of the heart upon the size of the fetus is of interest. The average weight of the children of the cardiac cases delivered at term and here reported was six pounds and ten ounces. This may be compared with the average weight at birth of seven and one-tenth pounds as given by Holt.

SUMMARY

Experience of two years with forty cases of chronic valvular disease in pregnancy has emphasized the importance of the following points:

1. An antenatal clinic is essential in detecting the early evidences of decompensation.

2. All cases showing decompensation in any period of pregnancy should be admitted to the hospital and given medical treatment, no matter what the stage of pregnancy.

3. Induction of labor should never be attempted in a case of decompensation until thorough trial has been made of medical measures.

4. The response to medical treatment of the average case of chronic valvular disease of the heart in pregnancy is satisfactory, and the same principles govern its treatment as govern the treatment of such cases not associated with pregnancy.

5. The termination of pregnancy in the presence of chronic valvular disease of the heart is not a matter about which hard and fast rules can be laid down. In general, if decompensation occurs early in pregnancy, or if it does not respond to medical treatment, if it has occurred and been severe despite proper care in previous pregnancies and if the signs and symptoms indicate serious lesion, termination is usually wise.

6. The method by which pregnancy should be terminated is largely an obstetric question, the point of greatest importance being the guarantee of a short and easy second stage.

SUMMARY OF CASES

1. Age twenty-six. Gravida 2. Admitted August 31, 1921. Complaint of headache and edema when admitted. Signs of mitral stenosis. Pulse rate 100. Delivered normally on day of admission. Discharged September 12, 1921, in good condition. Diagnosis: mitral stenosis.

2. Age twenty-two. Primipara. Admitted November 26, 1920. History of sore throats, scarlet fever and tonsillitis. Admitted with mild cardiac insufficiency. Signs of mitral stenosis. Delivered at term by Maurician extraction. Postnatal examination, June 14, 1921: No symptoms of heart weakness, slight hypertrophy; response to exercise, fair. Diagnosis: mitral stenosis, moderate hypertrophy, little dilatation. General outlook good.

3. Age thirty-seven. Gravida 3. Admitted December 13, 1919. Tonsillitis and arthritis in 1911. Symptoms of moderate cardiac insufficiency with signs of mitral stenosis. Blood pressure, 190/130; albuminuria. Improvement under treatment. Normal delivery at term six days after admission. Postnatal examination, June 14, 1921: Dyspnea on exertion, moderate hypertrophy of heart; blood pressure, 190/130. Diagnosis: essential hypertension, mitral stenosis; hypertrophy, slight dilatation of heart; toxemia of pregnancy.

4. Age thirty-three. Gravida 3. Admitted January 12, 1921. Pneumonia at 18 followed by heart trouble. Rheumatic fever at 13. Admitted with cardiac insufficiency, considerable hypertrophy and dilatation, and signs of mitral stenosis. Improvement under treatment. Delivery January 16th by medium forceps. Diagnosis: mitral stenosis. Outlook good.

5. Age thirty-one. Gravida 4. Admitted August 25, 1920. History of diphtheria and rheumatic fever. Cardiac symptoms for four years. Signs of mitral insufficiency on admission. Normal delivery on day of admission. Convalescence satisfactory. Diagnosis: mitral insufficiency.

6. Age twenty-six. Gravida 2. Admitted April 23, 1921. Past history negative. Dyspnea and signs of mitral insufficiency on admission. April 23, normal delivery. Postnatal examination June 28, 1921, showed the heart normal in size, position, and sounds. Diagnosis: mitral insufficiency, possibly relative.

7. Age twenty-three. Gravida 2. Admitted April 11, 1921. History of cardiac symptoms for several years. Signs of mitral insufficiency on admission but no signs of decompensation other than dyspnea on effort. Delivery was normal at term, April 11th. Postnatal examination July 5, 1921, showed no cardiac symptoms. A faint systolic murmur could be heard at the apex of the heart which was normal in size. Diagnosis: mitral insufficiency.

8. Age thirty-seven. Gravida 6. Admitted December 7, 1920. History of scarlet fever with kidney trouble at seven years, also a history of chorea and rheumatic fever. Threatened miscarriage three weeks before admission. Dyspnea with headache and edema and signs of mitral insufficiency and toxemia of pregnancy on admission. Blood pressure, 200/130; albuminuria, 100 per cent by volume. Labor was induced at the sixth month and a stillborn child delivered. Satisfactory convalescence. Diagnosis: mitral insufficiency; toxemia of pregnancy. At postnatal clinic, May 26, 1921, no complaint of cardiac symptoms was made. There was slight cyanosis and some edema of ankles, moderate hypertrophy and dilatation, signs of mitral insufficiency and hypertension. Blood pressure, 206/116. Outlook poor.

9. Age thirty-five. Gravida 4. Admitted January 29, 1920. History of influenza and malaria. Marked dyspnea and palpitation on admission. There were signs of mitral insufficiency. Blood pressure, 146/88. Labor was induced at the eighth month and a living child delivered. Condition on discharge was excellent. Diagnosis: mitral insufficiency. Examination in postnatal clinic, June 14, 1921, showed dyspnea, and edema of feet. There was obesity, the left ventricle was hypertrophied with signs of mitral insufficiency. Blood pressure was 180/110. Response to exercise was poor. Diagnosis: essential hypertension, obesity, relative mitral insufficiency. Outlook poor.

10. Age twenty-five. Gravida 3. Admitted August 28, 1920. History of tonsillitis and heart trouble. Eclampsia in 1917, since which, dyspnea, headaches and edema had been present. Admitted with signs of mitral insufficiency and toxemia of pregnancy. Blood pressure, 140/80; urine, albumin, 60 per cent by volume with casts. Five days later, delivery was normal after which the condition improved. Diagnosis: mitral insufficiency, toxemia of pregnancy.

11. Age thirty-nine. Gravida 7. Admitted October 12, 1920. History of heart trouble at 6 years and rheumatic fever in 1912. Admitted with edema, a trace of albumin in the urine, and signs of mitral insufficiency. Normal delivery on the day of admission. Diagnosis: mitral insufficiency, slight decompensation. Examination in postnatal clinic, June 14, 1921, showed moderate hypertrophy and signs of mitral insufficiency. No dyspnea except on unusual exertion or excitement. Outlook good.

12. Age thirty-two. Gravida 4. Admitted July 30, 1921. Moderate dyspnea, edema and cyanosis on admission. Very obese. In hospital nine weeks before labor, two weeks of which were spent in bed. Digitalis administered. Considerable hypertrophy but little dilatation; compensation fairly good. Normal delivery September 12. Condition improved. Diagnosis: mitral stenosis and insufficiency.

13. Age twenty-four. Primipara. Admitted February 3, 1920. Rheumatic fever at 12. At 21, was in the hospital because of heart condition. Admitted with marked decompensation, cardiac dilatation and hypertrophy, rate of 140, and signs of mitral stenosis and insufficiency. Moderate toxemia of pregnancy. De-

livery one week later at term by medium forceps. Child stillborn. Postnatal examination, May 26, 1921, poor response to exercise, marked hypertrophy, moderate dilatation. Diagnosis: mitral stenosis and insufficiency; toxemia of pregnancy. Outlook poor.

14. Age thirty-eight. Gravida 4. Admitted June 19, 1920. No antenatal care. Admitted in labor. Heart: hypertrophy and slight dilatation; some edema, infected teeth. Labor normal. June 28, 1921, at follow-up clinic, complained of dyspnea. The heart showed moderate hypertrophy and dilatation, mitral stenosis and insufficiency with impaired reserve power. Outlook only fair.

15. Age thirty-five. Gravida 2. Admitted July 12, 1921. Rheumatic fever as child. Latent cardiac trouble for years. No trouble with heart in labor nine years before. At sixth month of present pregnancy, had acute tonsillitis followed by palpitation, rapid, irregular heart action, dyspnea, nervousness. Slight hypertrophy, rate 130 with periods of grouped extrasystolic irregularities. Systolic and presystolic murmurs at apex. No stasis. Treatment: rest and digitalis with improvement. July 21, Cesarean section. Convalescence and eventual heart condition satisfactory. Diagnosis: mitral stenosis and insufficiency, acute infectious myocarditis following tonsillitis.

16. Age twenty-eight. Primipara. Admitted April 22, 1920. History of poliomyelitis at 2. Cardiac insufficiency for nine years, marked for past six months. Admitted with edema, albuminuria, and other evidence of pregnancy toxemia, mitral stenosis and insufficiency. Blood pressure 188/110. No improvement under treatment. Labor induced at the sixth month, four days after admission, after which, the heart became compensated. Diagnosis: mitral stenosis and insufficiency, secondary anemia, severe toxemia of pregnancy.

17. Age twenty-four. Primipara. Admitted February 17, 1921. Admitted with marked dyspnea, orthopnea, and edema. The heart was so over acting that no definite evidence of valvular lesion could be made out. There was albuminuria. Delivery, seventeen days later, was at term and normal, but during labor the pulse rate was 170 and the quality very poor. Compensation was restored soon after labor. Postnatal examination, June 14, 1921, showed cardiac hypertrophy of moderate grade. Diagnosis: mitral stenosis and insufficiency. Prognosis good.

18. Age twenty-eight. Gravida 3. Admitted July 10, 1920. Influenza and pneumonia in 1919. Earlier in pregnancy was admitted with dyspnea and edema, moderate hypertrophy of heart and congestion of lungs and was treated for fifteen days with improvement. Normal delivery on day of second admission. Convalescence satisfactory and condition good when discharged. Diagnosis: mitral stenosis and insufficiency.

19. Age thirty. Primipara. Admitted September 21, 1920. Rheumatic fever at 5 and at 10. Known cardiac disease since 12. In the ninth month of pregnancy, partial decompensation with marked hypertrophy and signs of mitral stenosis and insufficiency. Marked improvement after rest. Normal delivery September 28, 1920. Diagnosis: mitral stenosis and insufficiency.

20. Age twenty-one. Primipara. Admitted February 10, 1920. History of measles only. Admitted with marked dyspnea and enlargement of the heart, a rate of 120, and signs of mitral stenosis and insufficiency. Version and breech delivery at term on day of admission. Postnatal examination, June 28, 1921. Dyspnea on effort, marked hypertrophy and moderate dilatation. Diagnosis: mitral stenosis and insufficiency. Outlook fair.

21. Age twenty-eight. Gravida 2. Admitted February 26, 1921. Dyspnea and edema for two years, worse during pregnancy. Hemoglobin, 35 per cent. Ova of

uncinaria in stool. Heart: moderate hypertrophy and slight dilatation. Systolic and presystolic murmurs at apex. Spleen enlarged. Wassermann, ∓. Treatment: transfusion, digitalization, neosalvarsan. Delivery normal, March 6th. Much improved on leaving hospital. Diagnosis: mitral stenosis and insufficiency.

22. Age twenty-seven. Gravida 4. Admitted August 9, 1920. History of positive Wassermann reaction. Mild cardiac insufficiency for two months. Admitted with the signs of mitral stenosis and insufficiency of moderate degree, and of healed pulmonary tuberculosis. Delivery normal. Diagnosis: mitral stenosis and insufficiency.

23. Age twenty-eight. Gravida 2. Admitted March 15, 1920. History of rheumatic fever at 18, of repeated quinsy, of diphtheria and scarlet fever. Heart trouble for two years. Admitted with hypertrophy and dilatation, signs of mitral stenosis and insufficiency. Marked improvement followed treatment. April 2, normal delivery at term. Postnatal examination May 26, 1921. No symptoms of cardiac insufficiency. Diagnosis: mitral stenosis and insufficiency.

24. Age twenty-six. Gravida 2. Admitted September 26, 1921. Repeated tonsillitis. In Sloane Hospital in April, 1920, with toxemia of pregnancy. Labor induced. On admission, decompensated, showing dyspnea, cough, edema. In December, 1921, in hospital three weeks. Because of decompensation, abortion advised but refused. September 26, heart hypertrophied and moderately dilated, rate 100, regular. Presystolic and systolic murmurs at apex, stasis at bases of lungs, massive edema of legs and feet. Advancing decompensation. Death, September 28, 1921, from pulmonary edema.

25. Age twenty-seven. Primipara. Admitted May 25, 1920. Influenza in 1918. Severe toxemia with blood pressure, 225/120; albuminuria, edema, signs of mitral stenosis and insufficiency. Delivery the day following admission by forceps. Convulsion during labor and two afterwards. Full-term stillbirth. Diagnosis: mitral stenosis and insufficiency, eclamptic toxemia of pregnancy.

26. Age twenty-nine. Gravida 3. Admitted April 17, 1920. Past history included influenza and five attacks of rheumatic fever; also, diphtheria, scarlet fever, tonsillitis, and chorea. Marked cardiac insufficiency, hypertrophy and dilatation. Delivery, April 29th, was normal at term. Following delivery for ten days had Graham-Steell murmur. Discharged in good condition. Outlook satisfactory. Diagnosis: mitral stenosis and insufficiency, transitory relative pulmonary insufficiency.

27. Age twenty-seven. Gravida 2. Admitted March 9, 1920. History of rheumatic fever at sixteen years. Headache and slight cyanosis present but no dyspnea or edema. There were signs of mitral and aortic insufficiency with moderate enlargement of the left heart. Delivery was normal on the day of admission. Diagnosis: aortic and mitral insufficiency, mitral stenosis.

28. Age twenty-four. Gravida 1. Admitted March 21, 1920. History of tonsillitis, quinsy, and rheumatic fever. Cardiac symptoms for one year. Dyspnea, orthopnea, edema and some cyanosis on admission. Signs of aortic and mitral insufficiency with auricular fibrillation and a pulse rate averaging 100. There were a few coarse rales at the bases of the lungs. By medical treatment, compensation was restored in five days and the patient left the hospital returning in labor March 21st. Delivery was normal at term. Postnatal examination on June 14, 1920, revealed some cyanosis, a pulse rate of 88 and regular, marked hypertrophy and dilatation with general outlook poor. Diagnosis: mitral and aortic insufficiency.

29. Gravida 2. Admitted June 4, 1921. History of dyspnea, edema, and signs of mitral stenosis and insufficiency beginning with and continuing since first preg-

nancy. Labor, June 10, was terminated by forceps, eliminating the second stage. Postnatal examination, June 28, 1921, showed moderate hypertrophy and dilatation with excellent compensation. Diagnosis: mitral stenosis and aortic insufficiency.

30. Age twenty-one. Gravida 1. Admitted October 14, 1919. History of several attacks of rheumatic fever. Entered hospital with failing compensation, cough, and extreme edema. After six weeks of medical treatment and a trial labor of five hours, a cesarean section and sterilization were done. Convalescence was satisfactory. Diagnosis: mitral stenosis and insufficiency, possible aortic insufficiency, functional pulmonary leakage.

31. Age thirty-nine. Primipara. Admitted November 17, 1919. History of rheumatic fever nineteen years before. Admitted in labor with marked edema. Signs of mitral and aortic insufficiency. Labor was terminated by a medium forceps delivery November 18, 1919. Postnatal examination, May 26, 1921, showed marked hypertrophy, little dilatation and fairly good compensation. Diagnosis: mitral stenosis and insufficiency; aortic insufficiency.

32. Age thirty-nine. Gravida 6. Admitted March 17, 1920. History of measles. Slight cyanosis and edema on admission. The liver was palpable. Normal delivery at term ten days later. Condition satisfactory. Postnatal examination, May 26, 1921, revealed considerable hypertrophy but little dilatation. Diagnosis: mitral stenosis and aortic insufficiency.

33. Age thirty-eight. Gravida 10. Admitted April 17, 1920. History of subacute rheumatic fever. Dyspnea and marked edema for one month previous to admission. Signs of mitral stenosis and aortic insufficiency. Delivery was at term, April 8th, the child living about one hour. Diagnosis: mitral stenosis and insufficiency; aortic insufficiency.

34. Age twenty-eight. Primipara. Admitted September 27, 1920. History of diphtheria at two years, frequent colds and tonsillitis. No cardiac symptoms noted on admission. Delivery the day of admission was followed by cyanosis and signs of some enlargement of the right heart with auricular flutter. Eight hours later, the pulse was normal. Postnatal examination, May 26, 1921, revealed no heart lesion. Diagnosis: transitory dilatation of the heart with auricular flutter immediately postpartum.

35. Age twenty-four. Gravida 3. Admitted September 27, 1920. No history of previous illnesses. The physical examination was negative on admission. Immediately following delivery, breathing stopped and the heart became totally irregular with auricular fibrillation. In an hour the respirations were normal and the heart again regular. The patient left the hospital in good condition without evidence of valvular lesion. Diagnosis: transitory auricular fibrillation immediately postpartum.

36. Age forty-two. Gravida 13. Admitted May 3, 1921. Rheumatic fever seventeen years before. Moderate decompensation for two years. Heart showed dilatation and hypertrophy, rate 92, totally irregular with pulse deficit of 40. Systolic and diastolic murmurs at apex. Liver and lungs congested. Spent one month in bed with disappearance of cyanosis and edema and pulse deficit. July 14, reentered hospital in labor. Version under ether. Mother's condition good. Child died. July 21, 1921, compensation was good and patient's condition was much better than on May 3rd. Diagnosis: mitral stenosis and insufficiency; auricular fibrillation.

37. Age thirty-nine. Gravida 9. Admitted April 13, 1920. Rheumatic fever and long standing cardiac disease. Cerebral embolism one month before admission. Heart: moderate hypertrophy and dilatation, auricular fibrillation, congestion of

TABLE I.—SUMMARY OF CASES

LESIONS	NUMBER OF CASES	ANTECEDENT HISTORY				COMPLICATION		TREATMENT						RESULT		
		RHEUMATIC FEVER	TONSILLITIS	CHOREA	SCARLET FEVER	SECONDARY ANEMIA	TOXEMIA OF PREGNANCY	REST AND DIGITALIS	NORMAL LABOR	INSTRUMENTAL DELIVERY	THERAPEUTIC ABORTION	CESAREAN SECTION	STERILIZATION	AVERAGE WEIGHT OF CHILD	MATERNAL DEATHS	FETAL DEATHS
Mitral Stenosis, pure	4	2	1				3	3	2	2				6 lb. 10 oz.	0	1
Mitral Insufficiency, pure	7	3	1	1	1		3	3	5	2				6 lb. 14 oz.	0	0
Mitral Stenosis and Insufficiency	14	4	3		1	6	4	6	9	2	1	1	1	6 lb. 12½ oz.	1	4
Mitral Stenosis and Insufficiency and relative Pulmonary insufficiency	1	1	1	1	1			1	1					6 lb. 9 oz.	0	0
Aortic and Mitral Lesions	7	5	1					3	4	2		1	1	7 lb. 3 oz.	0	1
Auricular Flutter	1		1							1					0	0
Auricular Fibrillation No Valve Lesion	1								1						0	0
Auricular Fibrillation with Valve Lesion	3	2		1		1	2	3			3				0	3
Aortitis	1							1	1					6 lb. 11 oz.	0	0
Post-Infectious Cardiac Decompensation	1						1					1			1	1
TOTAL	40	17	8	3	3	7	13	20	23	9	4	3	2	6 lb. 10 oz.	2	10

liver and lungs. Incomplete left hemiplegia. Therapeutic abortion in third month. Diagnosis: mitral stenosis and insufficiency, auricular fibrillation, cerebral embolism. Postnatal examination, May 26, 1921, moderate dyspnea, slight awkwardness of left side, moderate hypertrophy of heart, with fibrillating auricle.

38. Age thirty-seven. Primipara. Admitted July 24, 1920. History of chorea and malaria. Cardiac insufficiency for six months becoming severe the last ten days. Therapeutic abortion, August 5, 1920, in the second month of pregnancy. Diagnosis: mitral stenosis and insufficiency, auricular fibrillation.

39. Age thirty-five. Gravida 2. Admitted March 25, 1920. Wassermann reaction, four plus. Moderate hypertrophy of heart with systolic murmur at base. No decompensation. Normal delivery, March 25th, at term. Postnatal examination, May 26, 1921: exertional dyspnea, edema, evidence of dilatation of the aortic arch and a systolic murmur at apex and over aortic area. Diagnosis: syphilitic aortitis. Outlook poor.

40. Age twenty-one. Primipara. Admitted March 11, 1921. Influenzal pneumonia three weeks before. Moribund on admission with extreme cardiac decompensation. Died shortly after admission. Postpartum cesarean section, child died. Diagnosis: postinfectious cardiac decompensation.

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(*For discussion, see page* 83)

EFFECT OF INJECTION OF EXTRACT OF ANTERIOR LOBE OF PITUITARY GLAND UPON THE ORGANS OF GENERATION*

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THE literature of recent years abounds with references to glands of internal secretion, and especially to the action of these glands upon the sex organs. Unfortunately, however, the contradictory findings of investigators in this field have left the subject in a state of uncertainty, so that we cannot as yet draw definite conclusions upon which to base therapeutic measures. In many instances, however, dogmatic statements bearing directly upon treatment have been made, although the experimental evidence upon which the statements are based is either incomplete or entirely unsatisfactory.

The importance of this comparatively new branch of medical research is such that every effort should be put forth to bring order into what is now unsettled and uncertain. Goetsch[1] from his study in this field concluded that: "All these developmental, structural, and functional changes in the sex glands of both the male and the female, produced by the feeding of pituitary extract, show an extremely selective and almost specific action of the latter upon the genital system. * * * The stimulating effect upon the sex glands is greater, the longer the influence of anterior-lobe administration is exerted." Goetsch and Cushing[2] found that: "Pituitary extract, and particularly extract of pars anterior, has a markedly stimulating effect upon the growth and development of the reproductive glands in young rats of both sexes, as evidenced by histologic examination." Uhlenhuth,[3] in a series of experiments on salamanders, found that the rate of growth of the animals fed on the anterior lobe of the hypophysis was greatly increased over the rate of growth of normal animals; and that this growth did not cease after they had reached the normal "maximum" size of the species, and experimental giants were produced. However, Sisson and Broyles[4] learned as a result of their studies that: "Feeding of the desiccated powder of the anterior lobe of the hypophysis of calves to albino rats from three to ten weeks of age in doses of 0.04 to 0.3 of a gm. causes no change in their normal development." Winternitz[5] fed dried extract of this gland to a group of chicks, running a second series as controls. He found that the hypophysis-fed chicks in one series laid eggs earlier and more frequently than the controls, and that

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after six months' feeding the pituitary fed group (consisting of 11 chicks) weighed five pounds more than the controls. This effect, however, could not be duplicated later and Winternitz says: "The marked

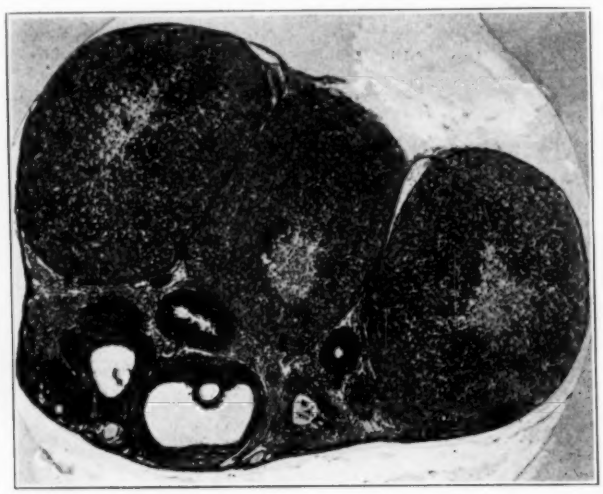

Fig. 1.—Ovary of rat, 11 weeks old, which had received daily for a period of 4 weeks subcutaneous injections of an aqueous extract of the anterior lobe of the pituitary gland.

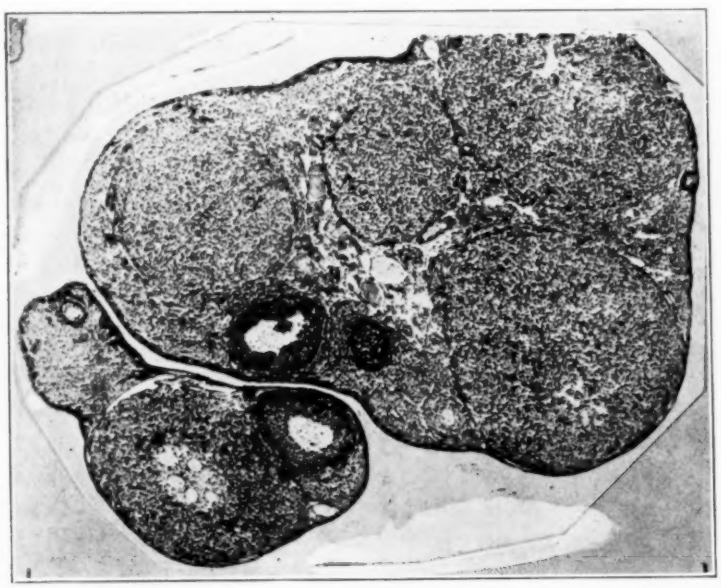

Fig. 2.—Ovary of rat, 11 weeks old, which had not received pituitary extract. Control to that shown in Fig. 1; same magnification.

changes apparently brought about by the administration of hypophysis in the first experiment is in great contrast to its inactivity in the subsequent ones, despite the fact that three separate brands of the

dried hypophysis, as well as the fresh glands, from the pig were used." Wulzen[6] maintained that the pituitary body, either injected or ingested, causes a diminution in rate of growth of young animals. Frank[7] after a most thorough and painstaking investigation concluded

Fig. 3.—Ovary of rat, 15 weeks old, which had received daily for a period of 8 weeks subcutaneous injections of an aqueous extract of the anterior lobe of the pituitary gland.

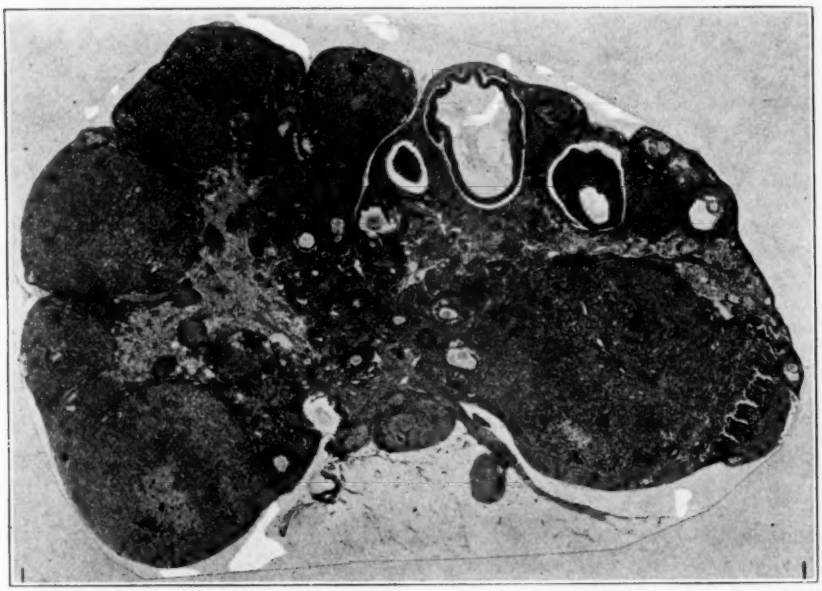

Fig. 4.—Ovary of rat, 15 weeks old, which had not received pituitary extract. Control to that shown in Fig. 3; same magnification.

that the feeding of anterior lobe extract composed of the lipoid fraction of the pituitary gland has absolutely no effect upon the sex organs of white rats.

Many more references could be cited, but the above are sufficient to show the present status of our knowledge and its contradictions.

Fig. 5.—Ovary of rat, 17 weeks old, which had received daily for a period of 10 weeks subcutaneous injections of an aqueous extract of the anterior lobe of the pituitary gland.

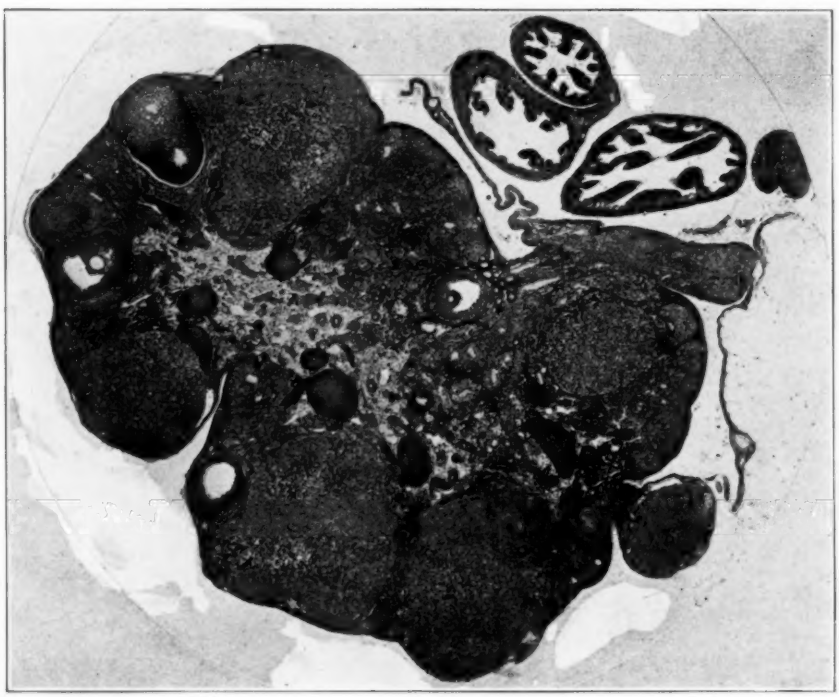

Fig. 6.—Ovary of rat, 17 weeks old, which had not received pituitary extract. Control to that shown in Fig 5; same magnification.

In this experiment the writer undertook to study the effects of injections of anterior lobe extracts of the pituitary gland upon the organs

of generation and the general growth development of the white rat. Since feeding the extract is attended by the possibility that (1) the animal might not consume and retain the entire dose, and (2) that digestive changes might render it inert, it was decided to administer the substance hypodermically.

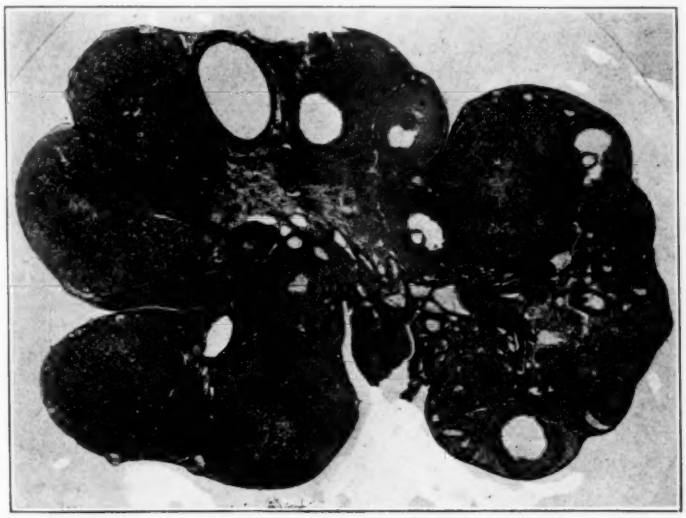

Fig. 7.—Ovary of rat, 22 weeks old, which had received daily for a period of 15 weeks subcutaneous injections.

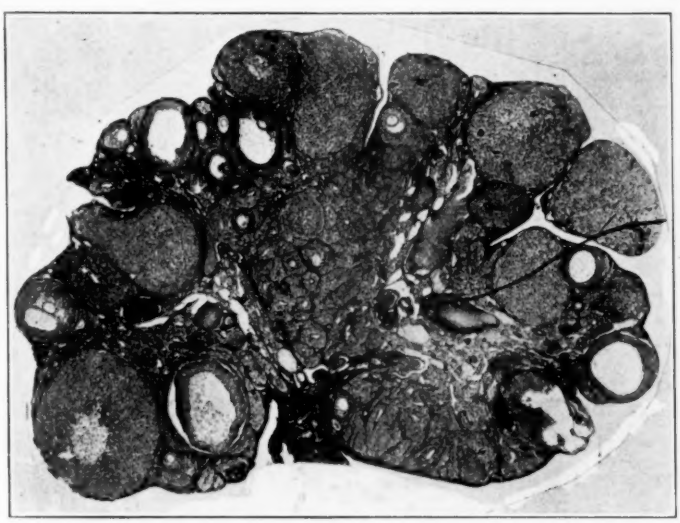

Fig. 8.—Ovary of rat, 22 weeks old, which had not received pituitary extract. Control to that shown in Fig. 7; same magnification.

Litters were bred from hardy laboratory stock of known pedigrees. Animals of the same sex in each litter were paired off. In each pair one was given the extract and the other was kept as control. Alto-

gether 52 animals were used. The material injected consisted of "antuitrin," a water-soluble solution of the anterior lobe of the hypophysis, prepared by Parke-Davis Co. The dose administered was 0.2 c.c., equivalent to 0.005 gm. of the desiccated powder. This amount was injected subcutaneously every day except Sundays. The animals were divided into three groups. The first consisted of 24 female rats—12 pairs of sisters; the second of 22 male rats—11 pairs of brothers. These two series were given the water soluble "antuitrin." A third group of 6 rats—3 pairs of sisters—was given the desiccated anterior

TABLE I

CONTROL

WEIGHT BEGINNING	END	GAIN
65	110	45
63	155	92
66	150	84
58	103	45
34	137	103
65	111	46
62	95	33
80	123	43
78	146	68
66	107	41
80	120	40
60	122	62
57	151	94
54	102	48
60	78	18
55	118	63
58	109	51
54	132	78
53	166	113
50	185	135
49	142	93
40	70	30
55	137	82
52	190	148
51	121	70
51	90	39

TABLE II

TEST

WEIGHT BEGINNING	END	GAIN
51	130	*79
45	104	59
65	145	80
58	95	37
30	122	92
64	96	32
60	86	26
80	88	8
66	125	59
62	95	33
60	101	*41
51	110	59
55	148	93
52	111	*59
58	102	*44
54	117	63
54	105	51
51	112	61
52	175	*123
45	190	*145
46	120	74
27	45	18
52	142	*90
44	150	116
49	107	58
31	60	29

lobe powder and was employed as an additional control. Wherever there was any difference in the weight of the animals in any pair, the larger one was used as control. The pituitary extract was administered for two weeks, the shortest period, up to fifteen weeks, the longest period. After the animals were killed, the genital organs, thyroid, hypophysis, and adrenal of each were removed and fixed in Zenker's solution, embedded in paraffin, and stained with eosin-hematoxylin. Serial sections were made of all ovaries, and while the entire uterus was not cut, large pieces, one taken from the middle of one horn and

another including the junction of both horns and their prolongations, were studied serially.

As a result of careful and detailed comparative study of organs from both the control and the test animals, the following can be said:

(1) Effect upon general growth and development. Tables I and II give the weights at the beginning and end of the experimental life period of both the control and the test animals. With few exceptions (marked with an asterisk) the pituitary-fed animals weighed not only no more, but even less than the controls.

(2) Effect upon genital tract, ovaries and uterus. In all cases the ovaries of the pituitary-fed animals showed the same or even less activity than the controls, possibly as the result of the frequent injections of foreign proteins. In none of the sections could any evidence be found to show an earlier sex maturity in the pituitary-fed animals as compared to the controls.

In view of these results, it can be stated definitely that extract of anterior pituitary lobe has no stimulating effect, either upon growth in general or upon the sexual maturity and activity of the organs of generation in the white rat.

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THE PHENOLTETRACHLORPHTHALEIN TEST FOR LIVER FUNCTION IN PREGNANCY*

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DURING pregnancy the normal physiological processes of the liver are augmented as a result of the greater metabolic activity of the pregnant woman, the building up of the fetal structures and the larger amount of waste material which must be detoxicated. Due to the demands of the fetus and the placenta the work of the liver in storing glycogen and in converting it into sugar is increased, for it has been shown by Slemons[1] that the supply of sugar to the fetus is brought about by a flow from a higher to a lower level, without any enzymatic activity on the part of the placenta, and consequently a higher level of blood sugar is necessary on the maternal side of the placental barrier. At the same time the placenta stores up glycogen as a reserve supply for the fetus. The liver regulating metabolism in general, is called upon to a great extent to detoxify the increased amounts of waste material resulting from fetal, as well as maternal, metabolism. The formation of urea from ammonia is another function of the liver which is increased during pregnancy for the same reason. The tendency to gain in weight in the pregnant woman is reflected in the fatty depots of the body, of which the liver is one. The relation of the liver to the formation of such substances in the blood as fibrinogen must be considered as being in excess of normal. Finally, the function of the liver in forming and excreting bile is probably less influenced than any of its other functions. That all these changes and increased demands throw a severe strain upon the liver of the pregnant woman is a patent fact.

Structurally the liver during pregnancy is enlarged, and is pushed upward backward and to the right by the growing uterus and its circulation thus somewhat impeded. In the liver of the pregnant woman fatty globules of varying size appear in the liver cells about the interlobular veins, this mild degree of fatty metamorphosis of the liver in pregnancy may be considered as a normal change. From this slight degree of fatty degeneration more progressive lesions may develop until an acute fatty degeneration with increase of the interlobular of fatty degeneration, accompanied by hemorrhage and thrombosis and atrophy, the picture may go on to an acute yellow atrophy. In

*Read before the John Morgan Society, April 7, 1922.

the various forms of toxemias of pregnancy, resulting fatally, marked pathological lesions of the liver have been noted. In the pernicious vomiting of early pregnancy the liver has been found to be the seat of an acute fatty degeneration, with a characteristic bulls-eye necrosis about the central vein. There is a marked resemblance to the pathology of acute yellow atrophy of the liver, and to the liver seen in fatal chloroform poisoning. In toxic albuminuria the liver shows a mild degeneration, parenchymatous in type, which, however, may be severe enough to present areas of necrosis and hemorrhage. The pathology of the liver in fatal cases of eclampsia is well known. Areas of hemorrhage of varying size are found under the capsule and on cut section are seen to extend at times deeply into the liver substance. There is an extreme fatty degeneration of the parenchyma, with areas of hemorrhagic and anemic necrosis. The focal necrosis about the portal veins is probably thrombotic in origin. In some toxemic conditions, occasionally seen in late pregnancy, with or without convulsions, there may be no albuminuria or other signs of renal insufficiency. These cases are usually fatal, show a tendency to deep coma, and present such signs of hepatic derangement as jaundice, and on autopsy marked autolysis of the liver function is found. The hepatic changes found at autopsy have led some French observers to consider them as the primary lesion of the disease, and to cause a fatal impairment of the liver function. The mere fact that the liver presents a pathological condition is not sufficient evidence on which to base the supposition that the cause is hepatic, and as in no other hepatic condition do convulsions occur, it may be assumed that the pathological findings in this organ are not the cause of eclampsia.

The frequent findings of pathological conditions of the liver in the toxemias of pregnancy have led many observers to undertake studies of the functional capacity of the liver in pregnancy. Thus far no one method has been found which would give sufficient evidence of a quantitative nature to allow a determination of the degree of injury to the liver. The discovery of such a method would assist in case of lowered ability of the liver during pregnancy in determining the time at which therapeutic abortion or induction of labor should be performed, and would serve as an index of treatment. Analyses of the urine of pregnant women have been carried out to ascertain the presence of urobilin and urobilinogen. Where other causes than pregnancy could be excluded, Litzenberg[2] has found these substances to be present in the urines of 25 per cent of pregnant women. This suggests the possibility of a "liver of pregnancy," probably due to deficient function, caused either by congestion, or by a toxemia too mild to be recognized in any other way. Other observers have found a somewhat lower percentage of urines of pregnant women showing

these substances. Urobilinogen occurs in the urine with very mild grades of liver injury, and its finding is specific proof of liver dysfunction, as the cells of no other organ are known to have the power of transforming urobilinogen into the other bile pigments. The mildly abnormal changes in the liver in pregnancy are believed to be responsible for the slight increase in the amino-acids in the blood of pregnant women, and in the similar increase in the amino-acid nitrogen in their urines. The hemoclasis crisis test of Widal has been applied in normal pregnancy, and has shown the protopexic power of the liver to be lowered in about a third of the cases tested, but the degree of leucopenia would indicate only a mild impairment of the liver. Titus and his coworkers[3] have utilized the estimation of blood sugar before and after the intravenous injection of glucose as a test of the carbohydrate storage ability of the liver cells, with varying results.

The successful use of the duodenal drainage tube with estimation of phenoltetrachlorphthalein elimination in the bile by Aaron[4] and others, has led me to apply this test to a series of pregnant women, with a view to determine the ability of the liver to eliminate the dye, the time necessary in normal pregnancy, and the degree of delay, if any, in clinically toxemic cases. The test has been performed as follows: The duodenal tube is passed on an empty fasting stomach, with the patient in the recumbent posture. After the tube has been swallowed to the duodenal mark the patient assumes the right lateral position to favor the passage of the leaden tip through the pylorus. It was found that the tip was in the duodenum in from one-half to two hours. The position of the tip in the duodenum is determined by the failure of water to return through the tube after its oral administration, the flow of golden, yellow, alkaline duodenal contents, not reacting to dimethylamidoazobenzol, and the position of air bubbles forced through the tube with a small syringe. If the tube was determined by these tests to be in the duodenum the patient was given 500 c.c. of water by mouth to establish a steady drip during the test, and an intraduodenal injection of 30 c.c. was made to promote the flow of bile. As soon as a steady yellow drip was present one c.c. of a solution of phenoltetrachlorphthalein, representing 50 mg. of the dye, was injected intravenously and the time of the injection noted. The duodenal contents were then collected in a series of test tubes, two minutes drip in each tube. To each of the tubes was then added one-half c.c. of a 40 per cent solution of sodium hydroxid, and the tube shaken. The first appearance of the dye is marked by a light brownish-pink color, which is soon seen to change to an intense purplish-red color. This first maximum appearance of the dye in the tubes is considered as the end-reaction, and the time of its appearance after the injection is noted. This same

intense purplish-red color will be seen in the remainder of the tubes, of which 24 are used.

Up to the present time twenty pregnant women have been tested by this method. The results are shown in the accompanying Table I. It will be seen that the time of the end-reaction varies in apparently normal cases from 16 to 24 minutes, with an average

TABLE I

	NAME	AGE	GRAV.	MONTH	END-REACTION	BLOOD PRES.	UROBILIN IN URINE	REMARKS
1.	M. R.	16	1	9	24 minutes	110-70	0	Normal
2.	M. H.	22	2	7	24 "	112-70	0	"
3.	S. A.	16	1	7	24 "	115-65	0	"
4.	H. T.	21	1	9	22 "	115-80	0	"
5.	L. G.	17	1	9	22 "	120-70	0	"
6.	G. S.	18	1	8	18 "	120-70	0	"
7.	M. P.	18	1	7	16 "	105-60	0	"
8.	E. B.	16	1	8	24 "	110-70	0	"
9.	M. P.	18	1	6	16 "	115-70	+	"
10.	F. S.	18	1	8	20 "	150-90	0	Albuminuria
11.	M. R.	19	1	9	16 "	110-85	0	Normal
12.	M. N.	20	1	9	18 "	120-70	0	"
13.	S. B.	15	1	7	20 "	90-50	0	"
14.	B. R.	32	3	9	20 "	125-85	0	"
15.	C. C.	22	2	9	22 "	110-70	0	"
16.	M. L.	35	5	7	16 "	96-60	++	"
17.	L. P.	40	3	1 d. pp	19 "	170-100	0	Nephritis, Convulsions
18.	V. W.	22	1	9	22 "	175-116	0	Nephritis
19.	B. E.	20	1	9	25 "	150-100	0	Albuminuria
20.	M. K.	36	9	1 d. pp	28 "	170-110	0	Albuminuria

of 20 minutes. In the cases of a clinically pathologic nature the end-reaction was variable. In one case where albuminuria and a high blood pressure were present throughout the pregnancy, and convulsions developed at the seventh month the end-reaction was reached in only 19 minutes. In this case the elimination of phenolsulphonephthalein in a three hour test was 10 per cent, 5 per cent, 3 per cent. In another case of albuminuria and a high blood pressure, with premature delivery of twins at the sixth month, following vigorous eliminative treatment, the elimination of the dye was delayed to 28 minutes.

SUMMARY

The functional activities of the liver are under a greater strain in the pregnant than in the nonpregnant state. The liver, under this augmentation of its functional capacity, undergoes a mild degree of fatty degeneration in normal pregnancy, which condition increases at times, under the various morbid conditions peculiar to pregnancy, to extreme degrees of degeneration, necrosis, hemorrhage and autolysis. The degenerations of the liver are probably secondary to the causes of the toxemias, and may be largely due to an exhaustion of its

glycogenic ability. The various tests for determining the functional ability of the liver in pregnancy are not sufficiently valuable from a quantitative standpoint. The phenoltetrachlorphthalein duodenal elimination time test has been performed on a number of normally pregnant women, with fairly constant end-reactions, and in several pathologic cases, with variable results where liver damage might reasonably be suspected to be present. Further studies will be necessary to properly evaluate this procedure.

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262 SOUTH TWENTY-FIRST STREET.

UTERINE POLYPS—HISTOLOGY, SYMPTOMATOLOGY AND A SUGGESTION AS TO ETIOLOGY*

BY SAMUEL H. GEIST, M.D., F.A.C.S., NEW YORK, N. Y.

UNDER the term uterine polyp is grouped a variety of lesions. They may exist as localized mucosal hypertrophies of polypoid structure, or may be definite tumor masses attached to the uterine or cervical wall by long thin or short thick pedicles, and covered by mucous membrane. They may project into the cavity of the fundus or cervix, protrude from the external os into the vagina, and in some instances, may even present in or project through the vulvar orifice.

There is however a great variation in the constituents of the polyps. They may be classified as adenomatous, fibromatous, fibromyomatous and angiomatous tumors. Under these types are found many variations in the histological elements composing the polyps.

Grossly, the form varies. In the body of the uterus, the adenomatous polyps are flat or rounded masses sometimes oval or lobulated. They are seldom larger than a walnut, though rarely they may grow to the size of a hen's egg. They are usually multiple, and occasionally may cover the entire endometrial cavity. It seems as if they have a predilection for the cornua of the uterus. These adenomatous polyps may become cystic, due to occlusion of the ducts and distention of the glands, and often the surface in this type may be pitted or irregular because of the rupture of the superficial cysts.

The adenomatous polyps may be sessile or pedunculated. The pedicle is usually short, not more than one-half of an inch, and usually rather slender. Rarely it may be elongated and the polyps project from the cervix. These growths whether they are pedunculated

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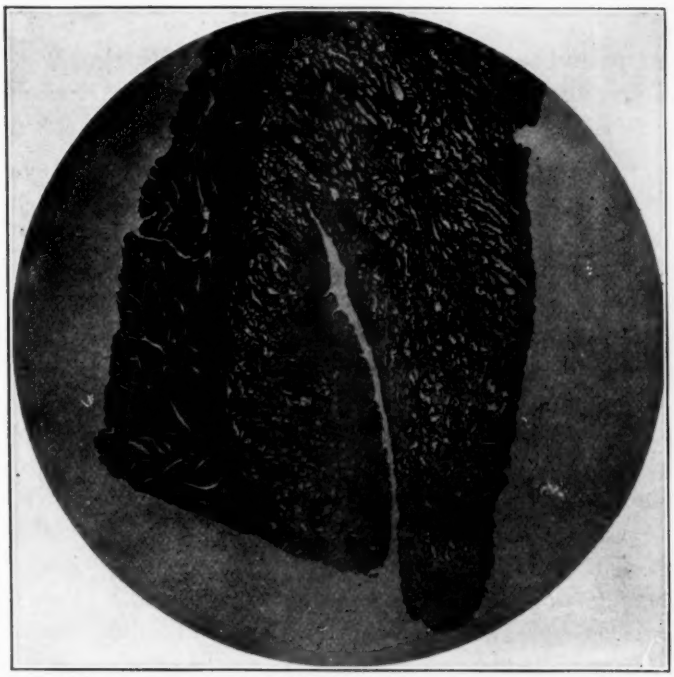

Fig. 1.—Fundal adenomatous polyp showing similarity in structure to the normal uterine mucosa.
Edema of stroma and dilatation of glands near tip.

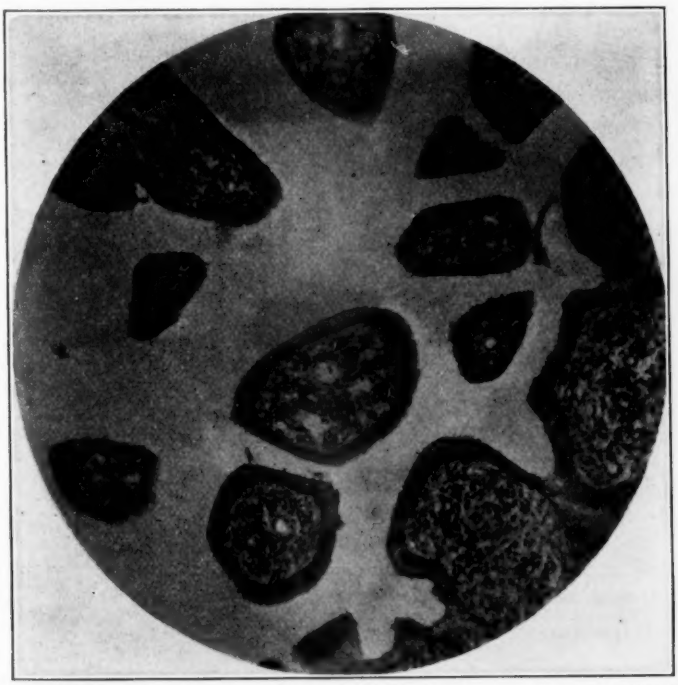

Fig. 2.—Papillary projections of polyp cut across showing round cells infiltrating a connective tissue core containing blood and lymph vessels and covered by high cylindrical (cervical epithelium).

or sessile merge into the surrounding mucosa without a distinct line of demarcation, and seem to be a part of the mucosa proper. (Fig. 1.) These masses are soft, velvety and occasionally show hemorrhagic infiltration in the tip, especially in the pedunculated variety.

In the cervix, the adenomatous type is rather common, in fact, there is a marked tendency for the cervical mucosa to form polyps especially in the presence of an inflammatory process. Here they are almost always pedunculated. In the cervix they may also be multiple. They are smaller, harder than in the corpus type, and due to the proximity to the external os, and to the tendency to form long thin pedicles, commonly project into the vagina. They are often cystic, the cysts being large and containing a thick mucinous fluid in contradistinction to the corpus polyps, which when cystic, are made up of tiny cysts containing a thin serous fluid. In some instances a nabothian follicle may be extruded and in the process present as a pedunculated cyst, i.e., a polyp. The cervical polyps are occasionally papillary, (Fig. 2), and the surface may show lacunae due to the rupture of the cysts, as is also true of the corporeal ones. Those polyps that project through the external os are almost invariably hemorrhagic due to interference with the thin-walled venous channels. Those that present in the vagina, even when not hemorrhagic, have a bright red velvety appearance, like the normal cervical mucosa.

The fibromatous or fibromyomatous polyps are really intramural fibromata or fibromyomata that are gradually extruded into the cavity of the uterus or cervix. They are hard, rounded tumor masses and vary in size from a bean to an orange. Depending on the stage of the extrusion they are either broadly sessile or pedunculated. The pedicles are firm thick strands, in contradistinction to the pedicles of the adenomatous ones, which are very soft. These tumors are often hemorrhagic and edematous with an eroded, ulcerated or necrotic surface due to the damage to the blood supply, which in this group is very poor and easily interfered with.

Histologically, the adenomatous polyps resemble the mucous membrane from which they arise. The gland content varies. In some instances the glands are extremely numerous with very little stroma while in others they are very scanty in number and show a corresponding increase in the stroma. The glands may be regular or irregular showing marked tortuosity, (Fig. 3), a racemose tendency, or they may be large, dilated, distended glands, cystic in character. This latter type is most common in the cervical polyps.

The epithelium covering the polyps and lining the glands shows variations in type. In the fundal type the cells are often high with granular cytoplasm and a small oval, or round, basally placed, pale staining nucleus. Occasionally in these fundal polyps ciliated cells are found on

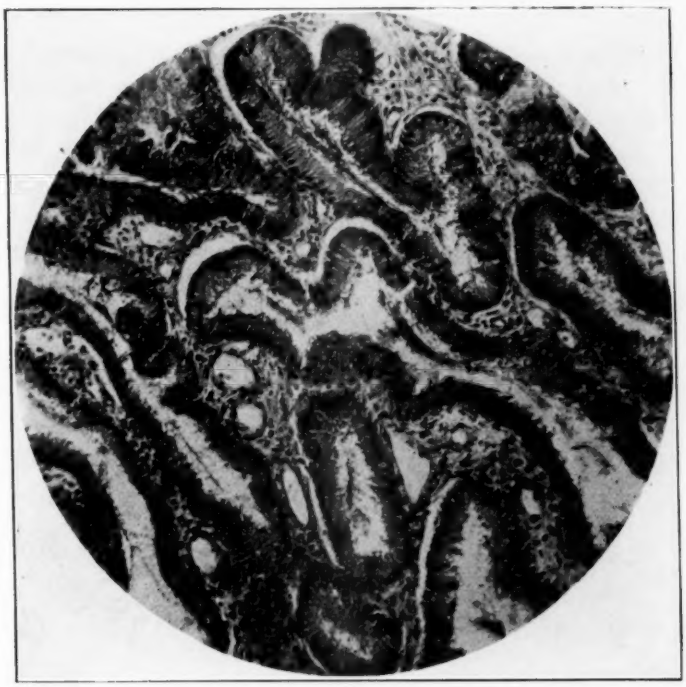

Fig. 3.—Markedly irregular tortuous glands with high cylindrical epithelium. *Not malignant.*

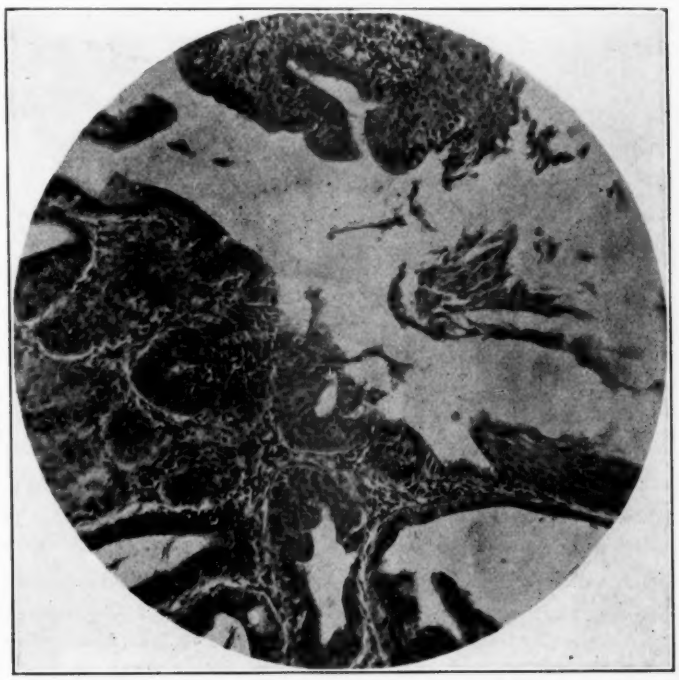

Fig. 4.—Section of polyp showing metaplasia with squamous epithelium extending into the glands. Not malignant. Uneventful postoperative course extending over a period of three years.

the surface epithelium. In the glands one may find multiple cell layers or rather a heaping of cells, of the same general character as the rest of the polyp, and with no suggestion of malignancy. One may also find areas of atypical cells consisting of two or three irregular rows. These cells may be of transitional type with a few high cylindrical cells on the surface and a more cuboidal type deeper in. These cell variations are not malignant but may be a point of origin for a possible malignant change. Occasionally there is a marked metaplasia in smaller or larger areas with typical stratified squamous epithelium. (Fig. 4.) This is especially true in the case of polypi projecting through the external os. Often these extruding polypi present papillary pro-

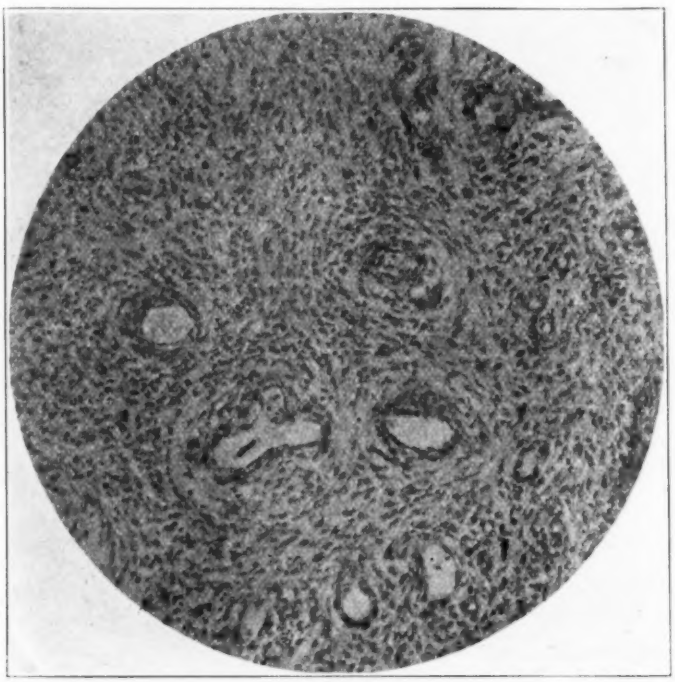

Fig. 5.—Numerous thin wall vessels in a cervical polyp.

jections (Fig. 2) composed of a fibrous tissue core containing small lymph and blood vessels covered by epithelium either stratified, transitional, cuboidal or cylindrical. The gland epithelium, too, may take part in these metamorphoses.

In the cervix the epithelium of the polyp is usually of cervical type, high cylindrical with a pale granular protoplasm and a basal oval nucleus. These cells resemble the typical mucin producing cells of the normal cervix, and the cysts which form more often than in the fundal type, contain a thick tenacious mucin. These cells, too, may present all the atypical variations mentioned above, as found in the

fundal polypi. The glands in the cervical polypi show a more marked tendency to become irregular and tortuous and the epithelium undergoes metaplasia more commonly than in the fundal types. It is essential to remember that many atypical variations can take place in the type of epithelium and in the character of the gland, without a true malignancy developing. The metaplastic areas may involve large areas of the polyp, the mass of glands may be tortuous and still the growth is benign. Mitoses are not numerous. Here, too, these areas may form a starting point for a malignant change, but infiltration into the surrounding tissue is essential for a definite diagnosis of malignancy. The metaplastic epithelium may extend under the more normal surface epithelium or even into the glands (Fig. 4), thus mimicking a true carcinoma. These metaplastic areas either on the surface or in the glands may be the starting point for a malignant growth.

The fibromatous or fibromyomatous polyps are really fibromata or fibromyomata, i.e., true tumors that are being extruded by the muscular contractions of the uterus. As they are forced into the cavity of the uterus they push before them the mucosa which then acts as a covering layer. As they extend into the cavity they lose their sessile character and become pedunculated. They are true tumors made up of pure fibrous tissue or fibrous and smooth muscle tissue. Occasionally one finds islands of tissue resembling the mucosa of the fundus scattered in the substance of the fibroid. The glands in these islands may be dilated and often contain old blood. Such a tumor is really an adenomyoma that has become pedunculated and is being expelled. The mucosa covering these tumors corresponds to the type that lines the cavity into which the tumor originally projected, i.e., either fundal or cervical. The pressure often, however, causes marked atrophy and one finds instead of a distinctive epithelial covering simply a layer of low cuboidal epithelium. In some instances even this disappears and there can be distinguished just a fibrous tissue layer and no epithelium. Often the surface is eroded and necrotic for the reason previously mentioned.

The stroma of the polyp resembles, as do the epithelial elements, the tissue of origin. In the fundal types it is cellular, made up of small round cells, with occasional lymphocytes or plasma cells and usually shows infiltration with leukocytes, of a chronic inflammatory nature. There are both lymph and blood vessels in the stroma, the latter being much more numerous (Fig. 5). These vessels are often dilated, thin wall sinuses, or sometimes rather thick walled veins and arterioles. Occasionally they rupture, especially in those polyps that protrude through the os, and thus produce a hemorrhagic blood infiltrated mass which may result in external hemorrhage. At times the vessels are so

numerous that the tumors are classified as telangiectatic polyps (Fig. 5) and may even resemble erectile tissue, when there is smooth muscle also present.

In the cervix the stroma is more fibrous in nature with areas of smooth muscle, inflammatory cells and vessels. Here especially is the richness in blood vessels marked, and in this type mechanical injury, torsion of the pedicle, or possibly the normal menstrual stimulation, may cause bleeding.

The pedicles of these protrusions may be short and thick or long and thin. The constituents are similar to those formed in the main mass, namely mucosa, either of cervical or fundal type, covering a mass of connective tissue, muscle tissue, vessels and glands, with usually a moderate inflammatory infiltration. At times, however, the pedicle may be so attenuated that it is represented by only a few fibrous connective tissue strands covered by an atrophic thinned out mucosa.

In the pedunculated fibromata or fibromyomata the pedicle is usually fairly thick and consists mainly of muscle, connective tissue, and blood vessels. In these tumors the pedicles may also elongate to an extreme degree and be represented by a few muscle or connective tissue strands covered by mucosa, and the tumor project through the cervix, where it may become infected, ulcerated, gangrenous or necrotic, due to twisting of the pedicle; or it may even slough off and lie free in the vagina.

The malignant changes that may take place in a polyp are of two kinds, either carcinomatous or sarcomatous. These are rare conditions but do occasionally occur. One must, however, distinguish the metaplasia that is fairly frequent, especially in polyps that project into the vagina, from a true malignancy. Metaplastic epithelium, in the form of squamous cells that may extend into the glands or under the superficial epithelium, must be distinguished from true malignancy with infiltration of the base of the tumor, and extension into the adjacent tissue, combined with other signs of malignancy, such as atypical cells, large deeply staining irregular nuclei and mitoses. Carcinoma may arise either from the surface epithelium or from the glandular epithelium and thus give origin to either a squamous cell carcinoma or an adenocarcinoma.

Sarcomatous change is much less frequent than the occasional carcinoma and may arise from any of the connective tissue structures in the growth. These changes are more frequent in the fibromatous or fibromyomatous polyps.

Symptomatically these polyps may not make themselves known. Many autopsies show uteri full of polyps both adenomatous or fibromyomatous, that give rise to no symptoms during life. The commonest

symptom, or rather associated condition, however, is bleeding. This symptom may take the form of a *menorrhagia* or *metrorrhagia*. At times it may be quite profuse, sufficient to cause a marked grade of anemia. The mechanism of this symptom is not easy to explain in all cases. It cannot be due to the presence of the polyp alone, for in many cases these tumors are associated with no symptoms and are purely accidental findings at autopsy.

In some of the cases associated with *metrorrhagia* the hemorrhage is due to an erosion of the tumor surface and so an injury to the vessels, or to a torsion of the pedicle and consequent rupture of the vessels. In almost all instances the tumors in these cases are long pedunculated ones that can be felt in the cervix or projecting through it. In other words, mechanical injury to the tumor surface or vessel, is the important factor.

In the group associated with menorrhagia there must be another explanation. Here the type of polyp is not the projecting long pedicled fibromatous or fibromyomatous tumor, but the small adenomatous type found in the fundus. Here, too, the mere presence of the abnormality cannot account for the bleeding as similar protrusions may occur with no symptoms. Also, the polyps are present between the periods and yet no bleeding occurs. It would then seem that the normal menstrual function is exaggerated in some of the cases associated with adenomatous polyps, and because of the rather frequent association it might be suggested that the two conditions, the adenomatous polyp and the bleeding, have a related etiology, possibly endocrine in origin.

Occasionally when a protruding polyp becomes infected there may be a foul purulent vaginal discharge present and if there is any absorption from an infected surface or tumor this may be indicated by a septic temperature. There is sometimes a *history* of dysmenorrhea, due possibly to the increased uterine contraction stimulated by the presence of the foreign body, and often there is a history of excruciating uterine pain when a tumor is being extruded.

On physical examination in the ordinary fundal type one can determine very little. Occasionally the uterus feels slightly enlarged and somewhat asymmetrical. With the pedunculated and extruding variety the examining fingers will detect a smaller or larger tumor, depending on the type, and of a consistency determined by the histology as mentioned above. Sometimes the small protrusions of adenomatous type are so soft that they are overlooked or rather not identified by the gloved finger, but the speculum demonstrates them as bright red, often very friable, vascular, protrusions. The ulcerated or necrotic ones present as inflamed reddish tumors, with a fibrinous or purulent

exudate covering part of the surface, and easily injured by the finger or an instrument, with resultant bleeding.

The question of the etiology of the polyps is a rather important one. It is true that in some instances inflammatory lesions may give rise to polypoid outgrowths of the mucosa, as for example the nasal polyp due to sinus disease. In cervical infections of long duration there is occasionally a hypertrophic condition of the mucosa, but in no instance does the hypertrophy take on the magnitude of the typical, so-called mucosal polyp. These inflammatory hypertrophies are circumscribed small masses varying from a pea to a bean in size. Another differential point is the presence of edema and marked inflammatory infiltration in the small hypertrophies associated with a chronic endocervicitis, and while areas of inflammatory cells are found in the real polyps they are by no means the predominating picture. Again the polyps are very common in the uterus and, as we know, real chronic endometritis is a rather rare condition. From these facts it seems rational to conclude that inflammation can only play a minor rôle in the causation of the mucosal polyps so commonly found.

There are several factors that would indicate that the polyps are simply local exuberant growths of the mucosa, especially those in the fundus, and that the etiological factor may be found not in the uterus but in some other organ, or as a disturbance of some normal physiologic function.

The hypertrophies partake of the histologic characteristics of the parent tissue (Fig. 1). There is no distinct line of demarkation from the surrounding tissue when examined microscopically (Fig. 1). The mucosa of the uterus merges directly into that of the polyp, streams in as it were, and the two tissues are almost identical in structure. True, there is a greater tendency to the formation of cystic glands, but that I believe is due to a mechanical interference with the emptying of the glands, especially those situated near the tip of the polyp. Again, the stage of the gland cycle in the polyp is the same as that of the mucosa of the uterus, and in those cases associated with bleeding, the histological picture is that of the hypertrophied mucosa that is so characteristic in cases of essential uterine hemorrhage or in the hemorrhages accompanying fibroids.

It seems that the so-called adenomatous polyps of the fundus are really not true tumors, but the products of some stimulation (endocrine). They only give rise to symptoms when there is real mechanical injury to them, except in those cases when the cause of the polyp, and not the polyp itself, gives rise to other symptoms, as hemorrhage. These polyps should not be classified as tumors.

In the cervix the same causative factor may be at work, but here inflammatory processes may give rise to polypoid mucosa hypertrophies

resembling the process in the nasal mucosa. The real tumors termed polyps, are more accurately described as pedunculated fibromata or fibromyomata, and symptoms associated with them are due to mechanical injury or to other extraneous factors.

We thus feel that we have to do with distinct processes included under the term uterine polyps, namely, real pedunculated tumors such as the fibromatous and fibromyomatous neoplasms, inflammatory mucosal reactions as seen in the cervix, and the localized mucosal hypertrophies so commonly found in the fundus. These latter are either associated with fibroids, with essential uterine hemorrhage, or exist as distinct entities without giving rise to any definite symptoms.

The one symptom most commonly associated with them is hemorrhage and, except for those instances of localized injury to the vascular channels, it would seem that the metro- or menorrhagia must have a factor other than the mucosal hypertrophy that determines it.

We believe after careful histological examination of those cases associated with atypical bleeding, that no lesion can be found to account for the hemorrhage except in the minority of cases and that the cause for this symptom is some factor not present in the tumor proper. As I have mentioned in previous publication, dealing with hemorrhage associated with fibroids, so-called essential uterine hemorrhage, and hemorrhage associated with ovarian and tubal tumors, the endocrine glands are most likely the important factor. Removing the ovaries by operation, x-ray or radium will cause these mucosal hypertrophies to atrophy or disappear, and will cure the associated bleeding. Because of this we must give consideration to the ovary as the factor of importance in the etiology of the symptom of hemorrhage, and probably for the presence of the hypertrophy. True, no histological lesion in the ovary can at the present time be demonstrated, but the disturbance is probably one of the normal physiology, that at least up to the present, cannot be determined. Again, the abnormal function of the ovary in itself may not be, and probably is not, the entire factor. The other glands of the known endocrine chain, and even the mucosa of the uterus, may by a perversion of their physiological functions act as important factors in the production of the pathological lesion in the mucosa, and of the abnormal bleeding.

I wish to thank Dr. J. Brettauer and Dr. F. S. Mandlebaum for the privilege of studying the pathologic material and the use of the clinical data. I am indebted to Dr. J. Globus for the photomicrographs.

300 CENTRAL PARK WEST.

SYPHILIS IN RELATION TO ABORTION, STILLBIRTHS AND INFANT MORTALITY*

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THIS paper is based on statistics obtained from the obstetrical and gynecological service of Professor B. P. Watson, in the Toronto General Hospital. Since February 1, 1918, Wassermann tests were performed on 1674 mothers at the time of delivery—of which number 73, or 4.3 per cent reacted positively.† These figures correspond very closely with those obtained in clinics elsewhere, allowing always for the presence of a negro population in certain of them.

No attempt will be made to pass judgment or to theorize upon the inconstancies of the Wassermann reaction. To look to the literature for light is only to be more confused, because of the diversity of opinions. It therefore has been taken as our method of diagnosis, fully realizing its shortcomings.

It is well known that certain fevers, such as pneumonia, and intoxications such as puerperal fever and eclampsia, as well as anesthesia often upset the reaction. Also, in the latter months of pregnancy the reaction is occasionally falsely positive.

Menten[1] among others, has shown that a positive reaction before parturition will often become negative shortly after delivery. In the series described the Wassermann was found in six cases to be negative on repetition after delivery, having been positive previously—the baby in each case being healthy and showing a negative test. Two of these women returned with second pregnancies and gave negative reactions. The babies again were healthy and their Wassermanns were negative. We have taken all six therefore, as examples of an upset due to pregnancy and classed these patients as nonsyphilitic.

Granting that with the above mentioned exceptions we are dealing with syphilitic women in the cases reacting positively, let us consider how great a factor is syphilis as a cause of stillbirth and infant mor-

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*Read before the Academy of Medicine, Toronto, January 5, 1922.

†These tests were made in the Serological Laboratory under the direction of Dr. H. K. Detweiler of the Department of Medicine, University of Toronto.

tality. Williams at Johns Hopkins, in a series of 10,000 cases records 705 deaths of children and estimates syphilis as the cause of 32 per cent of this total. Routh, after a lengthy review of vital statistics concludes that 20 per cent of the stillbirths and abortions occurring in England are due to syphilis and this, if correct, would represent a loss of 27,000 potential lives in England and Wales annually.

Deducting the six cases previously referred to, we have in our series, 67 women with definitely positive reactions. These women gave birth to 53 living babies and 14 stillbirths; approximately one stillbirth to every four living children.

Now before we follow the subsequent history of these children, may we consider for a moment the question of the fetal or cord Wassermann test? This is taken as a routine. As a diagnostic measure it is universally agreed that this is far from satisfactory and held by some to be so unreliable as not to be worth the time and expense entailed. Nevertheless, it does seem to portend to a certain degree the future of the newborn as the following figures show.

Fifty-three children were born to mothers with definitely positive reactions. Of these 28 gave positive cord Wassermann reactions, 21 were negative and in four the tests were not categorized. In two of these four the samples of blood were unfit for use and in the other two there was incomplete fixation.

Of the twenty-eight positive children 10 are still alive, that is at ages varying from a few months to between two and three years. Ten have died and eight have been lost to our records.

Of the twenty-one children with negative reactions thirteen are living, one has died and seven are off our records.

One of the four in which the reaction was indefinite is known to be alive, the other three we cannot trace.

Unfortunately, our "follow-up" system has in the past lacked proper organization and this accounts for the deficiencies in this report.

One case of interest is added to the above. The maternal Wassermann was negative, while the child, which died shortly after birth, gave a positive reaction.

The old law of Colles is familiar enough; namely, that a normal mother may give birth to a syphilitic child and yet remain immune to infection from it. This, of necessity, would indicate a direct paternal infection. Whether or not this explains this incongruous finding or whether the answer is that it is a result of a latent form of maternal syphilis, I cannot say. The question of the mode of infection of the ovum is still an open one.

There is a firmly established belief, which, however, of late has been questioned and seems to be disproved, namely, that syphilis is the most frequent cause of abortion or the termination of pregnancy in the earlier months.

The following are the figures obtained from a review of the cases of abortion in the Toronto General Hospital.

In 292 cases only 11 gave a positive Wassermann reaction, the remainder were negative, which is only 3.8 per cent specific.

A perusal of the records is rather illuminating in that we see 281 nonsyphilitic women having 1203 pregnancies showing a termination by abortion on 527 occasions; this is the appalling percentage of 43 per cent. It would seem, that the termination of pregnancy by unnatural means threatens the national birth rate to a far greater degree than we would ordinarily care to believe. Also, it is of interest to note that, the only three women giving a history of five or more abortions were all nonsyphilitic.

Adair[3] came to a similar conclusion with regard to the relationship between syphilis and abortion.

TABLE I

ABORTIONS

	NO.	%	AV. AGE	PREGNANCIES	ABORTIONS	% OF ABORTIONS	PARA ONE	MARRIED	5 OR MORE ABORTIONS
Syphilitic	11	3.8	28	44	15	34	1	2	0
Nonsyphilitic	281	96.2	29	1203	527	43	53	50	3
Total	292								

To return to the question of stillbirths, it may be stated that in 135 stillbirths, 14 occurred in mothers with positive Wassermann reactions.

Placenta previa and difficult labor are reported causes, both showing slightly higher figures than this and a much larger number of stillbirths occurred with no recorded cause, principally during the influenza epidemic of 1918.

This does not agree with the findings of other investigators, who would show syphilis as a far greater causal factor. For example, certain of the Continental workers would class 95 per cent of the macerated fetuses as syphilitic in origin. Our figures in no way approximate this.

To summarize our findings: In fifty pregnancies in syphilitic mothers, there were fourteen stillbirths and twelve deaths of the infant, yielding a birth rate loss of slightly over 50 per cent.

TABLE II

INFANT MORTALITY

	LIVING	DIED	NO RECORD
Mother positive—, baby positive at birth	10	10	8
Mother negative—, baby positive at birth	—	1	0
Mother positive—, baby negative at birth	13–x	1	7
Mother positive—, baby (?) at birth	1	—	3
	24	12	18

"x" Includes one baby who developed positive Wassermann which became negative on treating.

Note—Percentage of deaths 33⅓. Percentage of deaths with baby having positive Wasserman 54. The addition of 14 stillbirths shows a total infant mortality 52 per cent.

The masterful record of J. Whitridge Williams[4] lays great stress on the value of the correlation of the Wassermann reaction, the routine examination of placentae and the thorough postmortem examination of the fetus.

Some attempt has been made in our Department to study the placenta, but with little success; and the immensity of the work by a small staff is evident.

I have been able to find records of nine autopsies bearing on the question in point. Five stillborn fetuses were autopsied. In all these cases the maternal Wassermanns had been positive. In three the findings at autopsy were positive for syphilis. In one case the mother died of eclampsia. She had a positive Wassermann and it is now impossible to say whether this was due to her toxemia or an evidence of syphilis. The child in this case showed no evidence of syphilis, but in the liver, findings similar to the changes of eclampsia were present.

In four children autopsied the maternal Wassermann had been positive in three. The autopsy findings were positive in two. The other child had been born of a mother who had been under treatment for syphilis; her Wassermann was negative at the time of the birth and in this case the child showed no evidence of syphilis.

There are thirteen cases to report in which the mother had been treated previous to delivery. In seven of these the maternal Wassermann was still strongly positive at the time of birth. These mothers gave birth to six living children and one stillbirth. Three children died shortly after birth with very strongly positive reactions and three children survive, one of whom has a positive Wassermann and is under treatment. Three mothers gave weak positive reactions and their children are all living and apparently healthy. Three mothers gave negative Wassermann reactions. Two of the children from these mothers are alive and apparently healthy, the third child died a few

days after birth. At autopsy no evidence of syphilis was found. The same patient gave a history of two children who had previously died shortly after birth.

The following case is of interest. The mother gave a positive reaction. The baby gave a negative Wassermann reaction, becoming positive shortly after birth. Treatment was commenced and the baby is alive and apparently healthy. The mother, however, after two years' treatment still gave a positive reaction and upon becoming pregnant, gave birth to a stillborn baby.

In drawing our conclusions it is obvious that we cannot be dogmatic, but the following points may be made:

1. Mechanical interference is the most important factor in abortion as seen in a gynecological clinic.

2. That the relative importance of syphilis as a factor in the destruction of life increases with the period of the development of the pregnancy.

3. That even if the Wassermann reaction is not infallible it is a most valuable guide both to diagnosis and to prognosis.

4. The difficulty experienced in collecting the statistics of the small series reported only emphasizes the well-known need for close cooperation between the various departments and an efficient "follow-up" system.

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HYDATIFORM MOLE, WITH REPORT OF TEN CASES*

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THE rare occurrence of hydatiform mole is evident from the reports of recognized authorities. Edgar has seen it four times in 15,000 cases of labor observed in hospital and private practice and states that the condition tends to recur. He also quotes Mayer as reporting eleven recurrences in one patient. Pozzi (1909) stated that he had never seen an hydatid mole in over 6000 cases of labor observed in his hospital and private practice and Madame Boivin saw it once in 20,000 cases; Mayer (1911) reported one case in 310 and Donskoj (1911) stated that the incidence of hydatid mole at the Frauenklinik at Munich over a period of sixteen years was only one for every 4058 births. At the Harlem Hospital on our service we have seen six cases in 12,030 cases of labor and abortion, or about one in 2000, covering a period of eight years from January 1, 1914, to November 30, 1921. In addition to our six cases from the Harlem Hospital service we are able to add the reports of four cases, for three of which we are indebted to Dr. E. G. Langrock of this city, one being a private case of Dr. G. L. Brodhead.

PATHOLOGY

The nature of an hydatid mole has been the subject of much controversy; Virchow taught that they were simply myxomatous hypertrophied chorionic villi. Arthur W. Meyer in 1918 mentioned the importance of the work of Gierse published after his death by Meckle in 1847; Gierse claimed that definite transitional forms could be traced between the slightest change in the caliber of the villi and the most marked hydatid change. He described a villus showing hydatid degeneration from a chorionic vesicle about 12 mm. in size with the largest hydatid one-third of a line large; and further stated that such pathological changes are present in many abortions and seem to be the most frequent cause of abortion in the early weeks of pregnancy. While the work of Gierse has been either overlooked or disregarded by numerous writers and investigators and did not receive general recognition, his findings have been confirmed by Meyer's work at the Carnegie Institution in Washington.

Regarding the formation of chorioepithelioma following hydatid mole, quite a difference of opinion exists. Findley says that 16 per

*Read at a meeting of the Medical Association of the Greater City of New York, December 19, 1921.

cent of hydatid moles are followed by malignancy; Teacher believes that less than 5 per cent develop malignancy. DeLee states that chorioepithelioma (chorioma syncytioma malignum) gives a previous history of hydatid mole in about 50 per cent of cases, but he has had sixteen cases of mole none of which have developed metastases, nor have 75 others reported by König, Giglio and Kehrer. DeLee further states that the entire chorion may be changed or only a portion, leaving part of the placenta intact; pathologists, however, are not certain whether degeneration precedes the death of the fetus or follows it; probably both. Wood states that there are cases on record where a long period of latency (5 years) intervened between pregnancy and tumor formation.

INCIDENCE

In our ten cases three occurred between 20-25 years, three between 26-30; one between 31-35; two between 36-40; one between 41-45. The average age of these cases is 30.4 years, agreeing with the six cases of Poten with an average age of 32 years, ten of Donskoj at 25 years, twenty-three of Briggs at 28 years, six of Gromazki at 29.6 years and eight of Robertson at 28.4 years; also confirming the findings of Williamson who denied that hydatiform mole was especially common near the menopause.

PARITY

Three cases occurred in primiparae and seven in multiparae.

RACE

It is rather curious to observe that on a service which is composed of a greater proportion of negroes, that all of our six hospital cases occurred in white patients.

PERIOD OF GESTATION

One woman was two months pregnant; one, three months; four, four months; two, five months; one, five and one-half months; and one seven months. These statistics coincide with Meyer's statement that hydatiform degeneration is a common disease in the early weeks of pregnancy, being less frequent as term is approached.

APPARENT SIZE OF UTERUS

The patient who was pregnant two months showed an apparent size of two months, and the patient pregnant three months showed an apparent size of that period. The four women having a four months' period of gestation showed an apparent size of six months for two

cases, six and one-half months for the third case and seven and one-half months for the fourth case. Two women having a five months' period of gestation showed an apparent size of three months for one and five months for the other. Another patient five and one-half months pregnant showed an apparent size of three and one-half months while one who was seven months pregnant showed an apparent size of five months. This would tend to bear out the contention of Briggs that uterine enlargement in hydatiform mole is frequently not beyond the normal, which is contrary to the general belief.

WEIGHT OF MOLES

Unfortunately we were able to secure the weight of but five of our specimens, the largest of which weighed 1050 grams. This mole was extracted from a patient 40 years of age, a six para, period of gestation four months but with a uterus that had an apparent size of from seven and one-half to eight months. The red blood count after the manual extraction of the mole was 1,568,000, hemoglobin 30 per cent, and 900 c.c. of blood was given intravenously by the Unger method; two days later the red blood count was 2,240,000, hemoglobin 40 per cent; four days after this her hemoglobin had gone down to 30 per cent again and her red blood count to 2,096,000. The patient again received 800 c.c. of blood by transfusion; on the fourth day following this last transfusion her red blood count was 3,972,000, hemoglobin 65 per cent and she made an uneventful recovery. This patient has gained thirty pounds since she left the hospital in May, 1921. The other four moles weighed 896, 608, 384 and 256 grams, respectively.

TEMPERATURES BEFORE OPERATION

The temperatures before the delivery of the moles were as follows: Five patients had a normal temperature on admission and before delivery; one patient had a temperature ranging from 98.6° F. to 101° F. for two days before the extraction of the mole; one had a temperature of 100° F.; one had a 100.4° F.; one had 102° F.; and another had a temperature of 102.4° F. on admission, which rose to 104.4° F. before removal of the mole.

TEMPERATURES AFTER OPERATION

Three patients had a normal temperature after the extraction of the mole. Five patients had temperatures ranging from 101° F., to 103.4° F. for 24 to 72 hours after operation, returning to normal thereafter. One patient had a chill and temperature of 104.8° F. two hours after the extraction of the mole, becoming normal the following day. One patient, septic on admission, ran a temperature fluctuating between 102° F. to 104° F. for three weeks and then became normal.

URINARY EXAMINATIONS

The urine was examined in five of our ten cases. In four were revealed evidences of a nephritis, showing large amounts of albumin and many hyaline and granular casts; the other case showed a practically normal urine.

FETUS

DeLee (1918) states that ordinarily all traces of a fetus have disappeared but there are cases on record where a fetus has been present. In one of our series a perfectly formed four months' fetus was found. This is the only instance in hospital and private work, in our experience, where a fetus had been seen, and Edgar in a personal communication, states that he cannot recall a case where this occurred.

CLINICAL HISTORY

All of these patients gave a history of irregular bleeding without pain, or a serous or sanguineous discharge containing vesicles. The septic patient had bled for seven weeks prior to admission; in another instance the patient stated that the fetus had been discharged one month prior to admission; in another instance the patient had bled for two months prior to admission to the hospital; in only four of our cases was the enlargement of the uterus out of proportion to the period of pregnancy and curiously enough all of these women were apparently four months pregnant. In three of our cases the uterus was actually smaller than the supposed period of gestation. It is interesting to note that in three of our cases, hydatid mole was found in the first pregnancy. Four multiparae had had no previous miscarriage. One multipara had had one miscarriage seven years before, and two multiparae had had three previous miscarriages.

SUBSEQUENT HISTORY

All of the patients left the hospital in good condition within two weeks after removal of the mole with the exception of one woman who had a mild sepsis and remained three weeks in the hospital before her temperature returned to normal, when she also returned home in good condition.

We have endeavored to locate these patients in order to ascertain any subsequent developments and succeeded in getting in touch with six of them; they were all in excellent health and one (whose history I read before and who had passed a mole in May, 1921) has gained thirty pounds. The patient who became septic had a hysterectomy for uterine fibroids four months after leaving the hospital. She is now in good condition; no evidence of chorioepithelioma was found on pathological examination of her uterus. DeLee states that the

maternal mortality in hydatid mole is 13 per cent and gives the causes of death as hemorrhage, sepsis, uterine perforation and peritonitis.

TREATMENT

The treatment in all of these cases was manual removal of the mole where the mass had not been expelled spontaneously. We performed a digital curettage of the uterus; in some of the cases where we suspected the retention of some of the degenerated products of conception we first removed any débris with placental or sponge forceps before doing the digital curettage. The guarded use of a blunt curette may occasionally be necessary but we wish to caution against the use of a sharp curette on account of the extreme lack of resistance of the uterine musculature and the consequent danger of perforation. In all cases we were prepared to pack the uterus with iodoform gauze in the event of hemorrhage and inasmuch as hemorrhage is at times an alarming symptom it would be wise to make preparation for a transfusion.

CONCLUSIONS

1. That hydatid degeneration may occur at any age and at any period of pregnancy, being much more frequent however in the early months.

2. That on account of the toxic symptoms frequently shown the condition is easily mistaken for the toxemia of pregnancy until bleeding occurs and the appearance of vesicles clears up the diagnosis.

3. The uterus may be enlarged out of proportion to the period of gestation but as shown in our series of cases this is not the rule (four only showing such enlargement, while three were actually smaller).

4. These patients who have had hydatid mole should be kept under close observation for a period of years on account of the possibility of the development of chorioepithelioma.

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A CONSIDERATION OF THE PREGNANCY TOXEMIA KNOWN AS ECLAMPSIA*

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SPECULATIVE theories in medicine have had and always will have a prominent place in the art. Much that we have considered proved in the past and apparently clear enough in theory, turns out to be fallacious in practice and new lamps have been given for old in many instances, new lamps which have ceased to shine with the brilliancy they showed at the first lighting; and so we have, in part, at any rate, often returned to the scornfully discarded system for which they were exchanged.

Progress in the obstetric art, so-called, has been more marked in the last two decades than at any time in the history of medicine, but this advance has been so rapid, especially through the medium of surgery, that owing to the comparative safety of modern aseptic technic the surgeon rushes the patient to the operating table, many times it is feared, without due consideration of all the factors in the case. Far be it from the intention of the writer to belittle the marvelous strides and advantages of modern surgery, of which he is one of the most ardent and earnest advocates, but let us not forget in our enthusiastic moments that the *best* results in surgery are obtained by judicious operating at the right time in cases which will be *benefited* by the operation and not made worse. Babies were born, grew to mature estate, produced other babies and did surprisingly well both in their health and their duties to society in general, long before Lister discovered and gave to the profession the results of his theories which were so marvelous and far-reaching in their effects. True, of late years, we have reduced the mortality and morbidity of childbirth enormously in many directions, due to the knowledge and practical application of surgical principles to the subject in hand, but in other directions the improvement is not so marked if, indeed, any be noted at all, and it is of one of these instances that I wish to speak this evening.

Eclampsia, otherwise and more correctly known as the convulsive toxemia of the pregnant woman, has always been regarded as one of the most baffling conditions with which the physician has had to deal. Coming on with its lightning-like suddenness and, as was formerly supposed, in the absence of premonitory signs, signs which we have lately learned are present and usually can be recognized both by

*Read at a meeting of the Philadelphia Obstetrical Society, February 2, 1922.

means of better methods of diagnosis and a more accurate and thoughtful study of the complication, it was, and still is, enough to strike terror into the mind not only of the lay observer but also of the medical attendant and as with the introduction of modern surgical methods it became easier and more simple to empty the uterus with speed, it is small wonder that operative intervention in these cases became the accepted and approved method of handling them. Appearing in the pregnant state, what could be more natural and reasonable than the theory that the pregnancy must be the cause of the condition and that therefore to help the patient, the provoking cause must be removed, a theory which seems sound and to a certain extent undoubtedly is.

Whether or not, however, the practice in popular vogue is always the best for obtaining this result is the matter before us for consideration tonight and with your permission, I would like to take up the subject in the following order: 1. Frequency of occurrence of eclampsia; 2. the symptoms; 3. the pathology; 4. the treatments commonly in vogue both past and present, with the idea of placing the complication on a more rational basis.

Williams[1] in his text-book, considers that an eclampsia occurs once in 130 cases; Cragin[2] once in 79 cases and other authors in about the same proportion. At the New York Lying-In Hospital in 120,000 cases we have had 890 eclampsias. This shows that we may expect in hospital practice to see a case of this description once in about 185 patients. The season of the year apparently has some importance in this connection as the cases are noted more frequently in the early spring than at other times and also greater numbers are seen some years than others. Its occurrence is almost twice as common in primiparae as in multiparae (64.4 per cent respectively), and as might be expected much more frequent in antepartum than in postpartum or intrapartum patients.

The greatest number of cases are noted between the ages of 20 and 25 years which is in accord with the statement that the greater number occur in primiparae.

The symptoms may be divided into (a) the premonitory or pre-eclamptic, and (b) those occurring after the convulsive seizure has taken place. Of these the pre-eclamptic warnings are the most important, as like storm signals at sea, if properly noted in time, preparation for the approaching tempest can be made, and often a port reached, which will frequently though not always, as some authors have said, enable us to avoid the storm altogether.

It is difficult to put in the proper order of their importance the pre-eclamptic signs and symptoms, many authorities disagreeing markedly on this point, but to the reader it seems that by far the most constant and most significant warning of an impending toxemic state in the

pregnant woman, is found in the blood pressure. In an otherwise normal patient, a sudden and permanent rise in the blood pressure is to be looked on with alarm and is never of slight importance.

Next in order we have the examination of the urine, not so much from the standpoint of the condition of the kidneys themselves, as an index of the degree of toxemia from which the patient is suffering, and thirdly the results of the ophthalmoscopic examinations of the eyes, which as they are becoming more common seem to point out to us a very important piece of information. Edema of the extremities, or of the body in general, tenderness over the gall bladder, nasal hemorrhage, various digestive disturbances, constipation and so on, are undoubtedly of importance and when present should be carefully noted and if possible treated and corrected, but the increasing blood pressure, the urine examination both regarding the increase of albumin and the presence of casts and possibly the nitrogen coefficient (though this is of doubtful value) and the presence of progressive changes noted by the skilled use of the ophthalmoscope, are all that the skilled obstetrician needs to put him on his immediate guard for the onset of the eclamptic seizure. These when present, with the convulsion, the coma, the cyanosis, the rolling eyes, clenched teeth and other familiar features, need no further description, and we will now proceed to consider the pathology of the complication, in the light of what knowledge we have been able to accumulate during the last few years.

For a long time the disease was thought to be of renal origin, and as such was considered an uremia. More careful and extended observation, however, has shown that the kidney involvement is entirely secondary, as evinced by the fact that we frequently see cases in which, although typical in every other respect, clinically the kidney is not involved, or not until late.

The typical lesions found are those of a degeneration of the parenchymatous organs, notably the liver, followed by the kidney, less commonly by the spleen and pancreas; this takes the form of an albuminous change known as cloudy swelling, passing on to fatty degeneration early in the disease, and later in the more aggravated forms extending to all the tissues. In the other toxemias of the nonconvulsive type, we find zonal necroses in the liver lobules. These necroses have been described by Schmorl[3] of Germany and Williams[4] in this country.

In eclampsia, on the contrary, we usually find extensive hemorrhages in and about the portal spaces, with very little zonal necrosis in the outer space of the lobule. The hemorrhages are general in character, usually being especially marked in the brain, about the corpus striatum and pons, rupturing into the fourth ventricle.

Welch,[5] formerly pathologist to the New York Lying-In Hospital, explains these hemorrhages by suggesting that there is circulating

in the blood a poison which causes agglutination of the red cells, forming emboli, and then the solution of the endothelium of the blood vessels allowing an easy means of escape for the blood into the tissues. The blood pressure is usually high, especially during the convulsions, a condition which increases the brain hemorrhages occurring in these young subjects who are usually free from arteriosclerosis. Welch believed that the poison causing the intoxication is probably an enzyme or a combination of enzymes which attack the cells and cause their destruction, the process being known as autolysis; this statement is particularly applicable to the liver.

Concerning the immediate sources of the poison, they have been regarded as four in number: the food, fermentation in the intestine, cell metabolism, and the fetus and placenta, with a possible fifth in the kidney. It is a well-known fact that the autopsy reports almost invariably show dilated ureters in women dying in advanced pregnancy, and from retention there may be some contributory share. With regard to the part played by the fetus and placenta, little is yet known, and whether they furnish a portion of the enzymes which attack the maternal organism as has been declared by some, is a question which the biological chemist has not yet answered.

Hunter[6] states that eclampsia is due to a toxin, but what that toxin is has not yet been shown. McGarrison,[7] in his work on the thyroid, gives the following suggestion: Spinal anesthesia is produced by injecting a local anesthetic, such as stovaine, into the spinal canal, that is, bringing a paralyzing substance, into contact with the spinal cord. If instead of a paralyzing substance an irritant, like the toxin of eclampsia, is mingled with the cerebrospinal fluid, we would expect the results of irritation, namely, convulsions, as in eclampsia.

If it be true that the eclamptic toxin is to be found in the cerebrospinal fluid, it must get there through the choroid plexus which normally secretes the fluid. The choroid plexus is a true secreting gland, and in health has a selective action which prevents toxic bodies passing from the blood to the cerebrospinal fluid. If from disturbed function this selective action is lost, toxins will pass through the gland to the spinal cord, and those toxins may be the toxins of eclampsia. McGarrison mentions two classes of toxins: (1) Those resulting from endogenous metabolism and (2) toxins of bacterial action. Possibly the first class, by their presence in the cerebrospinal fluid, is responsible for the symptoms of eclampsia. The kidneys under normal conditions would secrete these toxins. In pregnancy they are much increased in quantity.

From McGarrison's[7] work it seems reasonable that the bacterial toxins may bring about a disordered condition of the thyroid, and that

they are originated in the alimentary canal. He mentions the frequency of the enlarged thyroid in pregnancy. The fetus causes increased activity of the thyroid which is more easily disturbed on account of acting under greater pressure. Most obstetricians believe that the toxin of eclampsia is elaborated in the alimentary canal of the mother.

McGarrison[7] concludes that the bacterial toxins elaborated in the alimentary canal are absorbed into the blood and carried to the thyroid apparatus upon which they act injuriously and cause insufficient hormone production. This leads to choroid plexus insufficiency with loss of selective action which permits (toxic) endogenous products of metabolism to enter the cerebrospinal fluid, where they act upon the central nervous system and produce the condition of eclampsia. If this view be correct, the rational treatment would be:

1. To remove the organisms which form the toxins from the blood.
2. To remove toxins, both bacterial and metabolic, from the blood.
3. To remove toxins from the spinal canal.
4. To supply hormones to activate the choroid plexus.
5. To treat symptoms as they arise.

LaVake[8] states that in the field of obstetrics there is not a more interesting or more important problem than that of the etiology of preeclamptic toxemia and eclampsia. He groups the principal theories into: 1. Bacterial; 2. Auto-intoxication; 3. Nephritic; 4. Liver; and 5. Ovular and placental.

The bacterial theory was presented as early as 1884. The main advocate of this theory has been Stroganoff, and his reasons for advocating are well worth citing: 1. General disease affecting all parenchymatous organs. 2. Acute infection commencing explosively or after a prodrome. 3. Fever accompanies it. 4. One attack confers immunity. 5. Marked genus epidemicus. In 1897 25 per cent died. In 1898 he had nineteen cases with a zero mortality. 6. It is impossible to explain the increase of eclampsia in populous centers otherwise than by accepting the infection theory. 7. As an argument against the uremic and fetal theories he mentions 126 cases, 10 of which occurred in the early months of pregnancy, and after the cessation of eclamptic seizures the pregnancy continued to normal termination. This could scarcely occur if eclampsia were due to toxins generated by the fetus. Early eclampsia usually affords the worst prognosis.

The auto-intoxication theory was brought forward by Bouchard, laying stress upon the toxins generated in intestinal stasis. He believes this view is tenable only from the standpoint of direct infection or absorption of toxic products resulting from the colon or other intestinal organisms.

The nephritic theory lays stress upon the presence of albuminuria and concomitant signs of nephritis with insufficient kidney function, the products of maternal or fetal metabolism being the offenders.

The liver theory accounts for the condition by the derangement of the liver structure and function as evidenced by anatomic and functional pathology.

The ovular and placental theories maintain the condition to be due to the generation of toxins from the products of conception or to infarcts of chorionic villi, and bring to their standard the force of the necessity of pregnancy in obtaining the condition.

LaVake states that in his experience the great majority of thromboses are caused by infection. For the past six years he has been interested in following cases with general pathologic conditions due to infections and trying to find the possible portal of entry. Obscure cases which clear up after the eradication of foci of infection are heard of every day. It has been proved that muscular pains have disappeared after the removal of infected teeth and tonsils. Cases of neuritis have improved under the same procedure.

Also, the author has been especially impressed by the cases of nephritis which clear up and remain so, after thorough eradication of dental and tonsillar infection.

Instances have been reported of eclampsia in the mother and nephritis in the child. Many have adduced from this that the products of the fetal metabolism causing the nephritis in the child were the cause of the eclampsia in the mother. It has been shown that organisms can pass from mother to child and it is possible that an infection was the cause of both conditions. Streptococci are the most common bacteria found in tooth and tonsillar infections, and their presence turns attention to those portals of entry in history-taking and in making complete physical examinations. The author calls attention to the absence of data regarding dental infection in many obstetrical histories and examinations. He presents 13 case histories of pre-eclamptic toxemia which came under his observation, cases which were typical and he dwells especially upon the histories of infection, where obtainable, and the evidence of infection. The data obtained in these patients point to the fact that it is important to eradicate foci of infection as soon as a case of pregnancy presents, or, if this is not possible, especial care should be exercised in determining the approach of toxemia. Every case should have a thorough dental examination and should be under the care of a dentist throughout pregnancy. The author disregards the belief of the laity and some of the medical profession that any dental procedure at this time is dangerous. He has never seen a case resulting in the disaster of an abortion from this cause and he has the assurance of many leading dentists that such an occurrence

has never come under their observation, although such men would undoubtedly use their judgment in avoiding long, tiring painful operations. The author concludes that adherence to the infection theory offers the best prospect of success in the prophylaxis of pre-eclamptic toxemia.

Thus it will be seen that while the etiology of the disease is by no means clear or settled, we still have made a marked advance over a few years ago, and are headed in the right direction. What the pathologist has done for us is to furnish the results of the findings post-mortem so arranged as to give us more definite ideas on which to work, and we believe that the time is not far distant when the actual toxin, enzyme or organism, which causes the complication and its method of operation, will be discovered.

Analyzing the situation, then, we are dealing with a woman who in the pre-eclamptic stage is just on the edge of being out of balance and who in the convulsive stage has lost her balance, so that in the first condition we must devote our efforts toward preventing the pathology from getting worse, thus allowing the convulsions to occur, and in the second to preventing them from recurring after they have occurred once, and incidentally only, of relieving the patient of the *cause* of her complication, namely the pregnancy; this latter, however, to be done without imposing on her a strain which will be greater than the one to which she is already being subjected.

I lay a great deal of stress on *the convulsions*, because I firmly believe that practically all of these cases die of brain hemorrhage due to rupture of the cerebral blood vessels, and that while it is not universally true that the higher pressure during the convulsion is the causative factor in the rupture of the vessel, it undoubtedly is the main one, as shown by treatment, of which more later.

It is acknowledged that we occasionally see cases which while otherwise presenting most of the features of the convulsive type pass away without any actual convulsions, but autopsy reveals the same brain hemorrhages as those present in the patients in whom convulsions did occur. These, however, are more profoundly toxic than the others and are apt to run a much higher continuous pressure which does not therefore affect our premise in the net result.

With these thoughts and facts before us then, let us proceed to take up the final subdivision of our subject, namely, the treatment. This may be divided into:

1st. Prophylaxis in the pre-eclamptic state.

2nd. Treatment of the actual condition when it has occurred. Both of these may be subdivided into (a) medical or conservative, and (b) surgical or radical.

Prophylaxis means watchful waiting. Watchful of small indicative symptoms, always suspicious that the blow is about to fall yet not rushing into operative procedures too hurriedly but waiting for a definite indication; in short, preparedness, in the most marked sense of the word. Every pregnant woman should be regarded as potentially pre-eclamptic, and that suspicion should not disappear until she has passed through her puerperium. Routine blood pressure, urine examinations and ophthalmoscopic inspections, together with general and special physical examinations should be made at frequent intervals, these to be increased generously if anything occurs which calls attention to any abnormal thing in the economy and it is only by this constant care that results will be obtained. It is often tiresome to physician and patient alike, especially to the latter, who cannot understand the necessity for such proceedings, and it is rarely wise to communicate to the patient the thought which makes us feel the desirability of these frequent visits. Still, the thing must be done, and faithfully, unless we are willing to take the chances of a severe catastrophe, which when in spite of these precautions occasionally does overtake us, is apt to reflect severely upon our professional judgment and skill, if all means possible for prevention have not been employed.

The diet should be regulated, meat and eggs largely discontinued, fluids given freely, proper exercise ordered and seeing that it is performed, sufficient sleep obtained and elimination by means of the bowels, bladder and skin secured. The teeth should be inspected and attended to. Printed instructions to patients are never given by the reader, as he prefers to consider each as an individual to be treated especially for the conditions suitable for her alone and not as one of a general class.

In the large majority of cases, if watched and handled in the above manner, no symptoms of toxemia will develop. Occasionally, however, in spite of this care, we shall one day, usually toward the latter part of the pregnancy, note a rise in blood pressure, possibly slight, possibly considerable, the urine may contain a trace of albumin and an occasional cast and eye changes may occur, slight but distinct.

We now come to the turn in the road. The signs point in two ways; shall we institute medical treatment and if so how rigid shall it be, or shall we terminate the pregnancy and if so by what means? What shall be the determining factor for the one or the other treatment? The writer confesses that he knows of no positive rule which should be applied in every case and the only thing that can be done is to be guided by one's experience and the judgment based on that experience. If rest in bed, low diet, possibly with an increase in the carbohydrate unit, elimination, etc., do not produce an amelioration of the symptoms or if they rapidly grow worse, termination of the pregnancy should be

considered before the patient has reached the point where the eclamptic seizures take place, and this termination if decided upon after such consideration, should be brought about by some one of the gentler means, in order to avoid the intense shock which otherwise may and usually does occur if the more forcible means are employed. Chloroform should never be used and rough manual dilation is absolutely not to be tried. Cases properly watched and cared for will rarely arrive at this stage, however, and we fortunately shall not often be called on to make the decision.

So much for the prophylaxis; we have seen that by its efficient use, the convulsive cases will be far less numerous than they have heretofore been. Nevertheless, there will always unavoidably remain the uncalculated few who, either from personal indifference or ignorance on their part, neglect on the part of the medical advisor, or a combination of both, or the very small proportion who though adequately and conscientiously cared for, still develop convulsions and who will demand all the resources we can bring to bear to suitably handle their complication.

As already stated at the beginning, the general feeling is still strong that the removal of the uterine contents is the most important step to be taken in the way of relief for the condition, and with the advent of modern surgical technic many operative procedures for this purpose have been employed. Rough manual dilatation followed by internal podalic version, with its resultant laceration and marked shock, vaginal hysterotomy, abdominal hysterotomy, have all been exploited and freely tried, and so firmly fixed are these maneuvers in the minds of most obstetricians that until comparatively recently they have been practically universally employed. The reader pleads guilty to having been one of the surgical enthusiasts until within the past five years.

Careful study of the results obtained both for mother and child, by these operative measures, compels thoughtful reflection, and when we consider that accouchement forcé give a maternal mortality of about 30.8 per cent, with an accompanying fetal mortality of 30-35 per cent, that cesarean section gives a high percentage of dead mothers with only a slightly improved fetal mortality, it makes one wonder whether or not these means of delivery are after all so efficacious as they would seem.

With this idea in mind, in 1916, the writer made up his mind to at least try out the Rotunda method of treating the actual convulsive cases, giving the method a fair trial, and to then compare the results with the operative method, with the idea of ascertaining if there was not some way in which at least an improvement in the maternal mortality could be made.

Since that time he has had 104 cases in which this method has been definitely followed; 17 mothers have died, or 16.3 per cent. Excluding those who were actually moribund when first seen, 7 in number, and in whom no treatment of any sort would have availed, this leaves a corrected mortality of 9.6 per cent for the net result, with a stillbirth mortality of 25.4 per cent. Certainly a startling difference when compared with the operative method which showed 30.8 per cent in 250 cases reported by the writer in 1909.

After this time a somewhat modified treatment was instituted which brought the mortality down to 17.4 per cent in 890 cases occurring in 120,000 confinements at the New York Lying-In Hospital up to January 1st, 1921. This, however, includes the author's 104 cases already mentioned which reduced the mortality figures considerably.

We now come to the conservative treatment so-called; this procedure was consistently carried out in all cases and unless the patients gave evidence of prompt delivery when the head reached the pelvic outlet, low forceps were permitted.

It is understood that all of the reported cases were pregnant or recently so; that they all had had one or more convulsions and represented true obstetrical convulsive toxemias.

Immediately on entrance to the hospital, the patient's blood pressure is taken, a catheterized specimen of urine secured, and she is put into an isolation room which is darkened and as much quiet as possible obtained. She is then given by hypodermic injection, one-half grain morphine sulphate, her stomach is washed out, two ounces of castor oil is poured down the tube at the end of the lavage, and she is given a colonic irrigation of five gallons of 5 per cent glucose solution.

If the blood pressure is over 175 systolic, phlebotomy is done, and a sufficient quantity of blood is extracted to bring the pressure down to 150; normal saline is not injected. In the experience of the writer, it is unwise to bleed the patient if the pressure is lower than 175 systolic, as if, for any reason, a good deal of blood is lost during the delivery, the pressure will be reduced so low that the patient may die from shock. The same objection applies to the *antepartum administration* of large doses of veratrum viride.

She is now kept quiet and one-fourth grain morphine administered every hour until the respirations drop to eight per minute. At this time convulsions have usually ceased, the patient will have fallen into labor, and, as has happened in practically all of our cases, will be delivered normally or by an easy low forceps in a short time. Occasionally the use of a little ether is necessary to control the convulsions while waiting for the effect of the morphine. The con-

valescence is treated in the usual manner, as indicated by the symptoms and has been in our patients significantly uncomplicated.

Summarizing our results, then, we see that:

1. The convulsive toxemia of pregnancy is a condition of whose exact cause we are unaware.

2. That the toxemia is divided into two groups: (a) The pre-eclamptic stage. (b) The stage of convulsive seizures.

3. That we can by careful watchfulness and intelligent supervision, largely prevent the condition from becoming severe, or from occurring at all.

4. That when it does occur, rough operative procedures do not give as satisfactory results either for mother or child, as does more conservative medical treatment judiciously combined with the gentler and less traumatic forms of operation.

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125 EAST THIRTY-NINTH STREET. (*For discussion, see page* 83)

BETTER OBSTETRICS AND THE PROBLEM OF THE BIRTH INJURIES OF THE NEWBORN INFANT*

By Hugo Ehrenfest, M.D., F.A.C.S., St. Louis, Mo.

FOR the past several years we have become fairly accustomed to have every writer and speaker in favor of "better obstetrics," refer to the appalling fact that "at least 15,000 women die every year in the United States from conditions, almost entirely preventable, caused by childbirth." The practically exclusive use of this one argument would seem to imply that conditions are entirely satisfactory as far as the newborn child is concerned. As a matter of fact they are not. It now is well established that approximately in 40 per cent of necropsies, performed on all stillborn infants and those dying within the first few days of life, definite traumatic intracranial lesions can be discovered, that roughly in about one-half of the cases these lesions are directly responsible for death, and that in the other half they represent more or less incidental findings though always proving the actual traumatization of intracranial structures during birth.

This newer information, in my belief, has not been sufficiently broadcasted among the very many physicians who are attending women in labor. Even expert obstetricians appear to be slow in appreciating their own obligations towards this problem of parturitional traumatization of the infant, and in recognizing the relation of this problem to that "better" obstetrical care which must be furnished to the women as well as to the newborn babies of this country.

Speaking here to a select group of experts I take it for granted that you are acquainted with this striking frequency of intracranial birth traumatisms so often emphasized in recent literature. You possibly are not aware of the fact that a more complete examination of the body at autopsy, a routine which includes the study of the vertebral column and a macroscopic and microscopic study of all abdominal viscera, reveals in a large percentage of the cases (exact figures are not yet available) also vertebral fractures, especially in the cervical section, or hemorrhages in liver, pancreas and very commonly in the suprarenal glands—all of them seemingly of traumatic origin.

Many entirely unsuspected birth injuries are discovered in a routine examination of every infant immediately after birth, if only more carefully done than is the prevailing custom, especially if such a

*Read at a meeting of the St. Louis Gynecological Society, February 23, 1922.

routine includes in every instance the palpation of the entire skeleton, an ophthalmoscopic examination, observation of the behavior of the child for the first few days of life and, in selected cases, in which some injury is suspected, an x-ray examination or a spinal puncture. A routine of this kind, here and there adopted in large maternity services, has led to the discovery of many important facts of which I shall mention but a few. Evidences of a retinal hemorrhage can be seen in about 20 per cent of normal sized infants born after normal spontaneous labors. This percentage of retinal hemorrhages rises to 40 in premature infants, and to 50 for babies born through contracted pelves. Probably in about 10 per cent of the cases a forceps extraction is followed by a facial palsy. Facial palsies are not exceptionally rare even after labors terminated spontaneously. The actual incidence of clavicular fractures is probably close to one and a half per cent of all deliveries, being apparently a little over 8 per cent for forceps extractions and around 6 per cent after version followed by extraction. I fear no contradiction in asserting that in the large majority of instances the attending accoucheur remains unaware of this injury to the clavicle. In most of the relatively few cases in which the damage is noticed at all, the discovery is made by nurse or mother if a larger callus forms.

Such a systematic search for injuries of the newborn has proved not only that traumatizations of all kind are fairly common but also that in many instances they are due to a brusque manipulation in the attempt to resuscitate the asphyxiated or seemingly asphyxiated newborn. In this manner are produced many slighter and severer lesions in the mouth, on palate and tongue, pulmonary injuries by means of the various pumps, clavicular fractures, shoulder injuries, fractures of ribs and, as irrefutably shown, intracranial lesions, vertebral fractures and liver ruptures. A simple consideration would seem to lead to the inevitable conclusion that swinging of the child, forcible bending and extension, or its mere suspension by the legs with the head hanging downwards, in the presence even of only a slight intracephalic traumatization, must necessarily tend to increase the hemorrhage from an injured vessel, and especially so if the newborn's blood, as often is the case, exhibits an abnormal delay in clotting time. Incidentally I may point to the fact that the very traumatization of the child in birth will produce a clinical picture closely resembling that of deep asphyxiation, which, if not properly interpreted, necessarily leads to additional traumatization by the efforts of resuscitation. Among others, it was this reflection which prompted me to suggest the following systematization of all the many different causes of intracranial birth injuries advanced by various writers. The primary causes, possibly without exception, are of a mechanical nature. Intra-

cranial structures are damaged by the forcible, excessive or, especially, by the quick compression of the head. For the majority of these cases the responsible mechanical cause can be recognized in definite anomalies of labor, difficult forceps extractions, breech labors, difficult extraction of the aftercoming head, etc. There still remains a fairly large group of fatal intracranial injuries in which labor was easy, spontaneous, often precipitate, the child small, frequently premature. Also in these cases the immediate cause of the injury is a mechanical one—the compression of the head in its passage through the birth canal, both through its bony and its soft portions. We find that in these instances asphyxiation and, most of all, prematurity had rendered the infant abnormally susceptible to the trauma even of a seemingly normal labor, and must conclude that these conditions have to be regarded *predisposing causes* in the etiology of intracephalic birth injuries. In the presence of only slight intracranial lesions, a decreased coagulability of the blood and inappropriate manipulations during resuscitation necessarily tend to hasten, or to prolong, the escape of blood from injured vessels, though small, and thus hemorrhagic diathesis and all violent maneuvers during resuscitation represent definite *contributary factors* in the causation of cerebral hemorrhages.*

After this digression I must return to the facts revealed by the systematic study of all newborn infants. Most important among them is the knowledge that a large number of children sustain in birth and, more often than generally appreciated, in the attempts to stimulate suspended respiration, injuries which do not prove fatal.

This, in my belief, is a most noteworthy fact. Obviously it suggests the question: "Are such injuries, at least in some cases, responsible for certain physical and mental defects manifesting themselves only later in life?" This is not a new question. Indeed, some time ago the neurologist answered it emphatically—too emphatically in my belief—in the affirmative. It was the neurologist who told the obstetrician that a "difficult" labor—whatever this term means—is an important etiological factor in the causation of feeblemindedness, idiocy, epilepsy, speech defects and so on. More recently, obstetricians have exhibited an alarming willingness to accept the judgment of the neurologist, and we now hear from the obstetrician, and read in some textbooks of obstetrics, a good deal concerning the necessity of shortening labor by means of pituitary extract, by forceps extraction, or cesarean section in order to protect the child against an injury of its brain. Personally a most careful critical study of the problem has convinced me that the dreaded long continued compression of the fetal head proves decidedly less dangerous than its quick compression

*See, Ehrenfest, Birth Injuries of the Child, D. Appleton and Co., 1922.

by a forceps extraction or as the result of too large a dose of pituitrin. I cannot claim that my own conclusions necessarily are correct. This problem requires further study—by the obstetrician and not the neurologist.

Systematic studies, diligently made in large maternities, have, as already pointed out, established the fact that nonfatal intracranial lesions, retinal hemorrhages, fractures of various bones, epiphyseal detachments, and dislocations of certain joints are more common than obstetricians suspect or textbooks mention. Therefore, I also have come to the conclusion that many conditions, today somewhat indiscriminately termed congenital, simply because they existed since birth, really represent birth injuries; while again others, as e.g., torticollis, are not birth injuries, as commonly claimed, but represent truly congenital anomalies. This means anomalies of development during intrauterine life, as for instance now generally conceded for the hip joint dislocation, which not so long ago was still charged up against the obstetrician.

The obligation of the obstetrician towards the momentous problem of birth injuries of the child, as I see it, is twofold. He is obviously interested in the question of their successful prevention. He seems less aware of his great responsibility for their early discovery and accurate diagnosis.

In regard to prophylaxis the assertion can be made that, while certainly not all traumatization can actually be avoided, the incidence of the more serious injuries decreases more or less proportionately to greater expertness of the attending accoucheur. This expertness does not imply solely greater mechanical skill in the execution of obstetrical manipulations and operations, but also a diagnostic ability in recognizing minor anomalies in the configuration of the pelvis or in the mechanism of labor, and, finally, that undefinable quality of sound judgment in the decision whether or whether not, and at what moment, interference is necessary and likely to prove advantageous. The problem of prophylaxis has been rendered decidedly more complex by the recent knowledge that many of the serious and slighter traumatisms of the newborn are seen subsequent to spontaneous and quick labors, especially, of premature infants. In about 20 per cent of intracranial lesions, discovered at necropsy, the history shows that the labor had not been terminated by artificial means or operations but was normal in every respect. This newer knowledge has lessened but not eliminated that responsibility with which the obstetrician used to be charged for such injuries.

This again is a fact deserving emphasis. The obstetrician should show much less hesitancy, than in the past, to make openly the diagnosis of a birth traumatization. A more fearless attitude, in my be-

lief, would prove of incalculable value in the desirable improvement as regards the second relation of the obstetrician to birth injuries, namely, his responsibility for their prompt and correct recognition. This holds particularly true as far as the intracranial lesions are concerned.

The admittedly unsatisfactory results of their treatment, surgical and otherwise, in the main are due to the prevailing custom of not calling a consulting pediatrician or surgeon until the infant has general convulsions or is comatose, i.e., at a time when the infant is in a well-nigh hopeless condition, an extremely poor surgical risk, with the surgeon looking in vain for any symptoms that might offer a clue for a more exact localization of the hematoma. As a matter of fact, the very first signs of the intracephalic injury often are insignificant, at times not immediately noticeable. There can be no doubt that a more careful observation on the part of the obstetrician, who under prevailing conditions is the only one who sees the baby immediately after birth, would reveal in many instances a number of symptoms which commonly precede the first convulsion or the stupor. I cannot attempt to enter here into a thorough discussion of the early symptomatology of intracranial injuries and shall but briefly mention a few of these symptoms, recognizable only by careful study, occasionally of but short duration: A spasticity limited to one extremity; the extension of this spasticity to another extremity; the unilaterality of symptoms; the delayed involvement of cranial nerves; or a sequence in the development of these symptoms which occasionally, exactly like in an experiment, accurately indicates the primary focus of the hematoma and its gradual extension in a certain direction within the skull. An immediate ophthalmoscopical examination, a study of the blood clotting time, and a prompt spinal puncture furthermore are diagnostic, and the latter incidentally also therapeutic, procedures of an importance which is not as yet properly appreciated by the obstetrician.

Careful examination and observation of every newborn child, particularly when including an eye examination, will reveal in many cases a marked spasticity in some muscle groups, a paralytic or paretic condition in others, slight convulsions or retinal hemorrhages, all symptoms that might disappear more or less promptly and by their very evanescence will suggest that the infant apparently is in a perfectly normal condition. A continued observation of this group of infants, extending into later childhood or even into adult life—obviously to be made by the obstetrician who alone had noticed these symptoms—is likely to enable the obstetrician some day to solve authoritatively the problem of the truly congenital or traumatic origin of certain pathological conditions, and to furnish a definite answer to the mooted

question of the etiological importance or unimportance of particular traumatizations in birth for physical and mental defects manifesting themselves only later in life.

I maintain that today the obstetrician is dereliet in his duties towards the newborn child. As long as the conditions now prevailing in the practice of obstetrics force him to take sole responsibility for the newborn, or as long as unjustifiable jealousy and unwarranted pride prompt him to bar the pediatrician from the newborns' ward of the obstetrical department, he will have to exhibit more interest in the baby, at least during the first few days of life. I can assure the obstetrician that a keener appreciation of this obligation will not prove a task only onerous. He will discover, I venture to say, that the ever growing tendency to hasten labor or to make it a less painful procedure, is not entirely compatible with the best interests of the child. He will experience the satisfaction of his increased usefulness both to mother and child by not only preventing more successfully parturitional injuries of the infant, but also by protecting the child against the immediate or delayed consequences of such injuries, whenever he has recognized them promptly. To you, as expert obstetricians, I wish to point out the wide and virgin field for many interesting and valuable observations and investigations which offer fair promise for enrichment in our still limited information concerning the etiology, diagnosis and prognosis of the birth injuries of the child. The existing deficiency of knowledge of this vast and important problem can justly be charged against us obstetricians, who have persistently failed to study more carefully the newborn, and at the same time have, more or less deliberately, prevented the pediatrician, neurologist and surgeon from acquainting themselves with the behavior of the normal and abnormal newborn immediately after birth.

METROPOLITAN BUILDING.

(*For discussion, see page* 99)

ORGANIC DISEASES OF THE NERVOUS SYSTEM COMPLICATING PREGNANCY: WITH A REPORT OF TWO CASES*

By Edward A. Schumann, M.D., and Harry S. Fist, M.D.,
Philadelphia, Pa.

ORGANIC disease of the nervous system, coincident with and complicating pregnancy and labor, is fortunately an event of rare occurrence. In reviewing the literature, however, one is struck by the comparatively large number of reported cases, the lesions ranging from mild postpartum neuritis or pressure palsies to complete transverse myelitis with its usual fatal termination.

Two cases recently observed by one of us, have led to a survey of the subject especially from the standpoint of the relationship of pregnancy and nervous disease with respect to etiology. The field is an enormous one and there has been no attempt made to study it in its entirety, but two varieties of disease being considered, puerperal neuritis and acute transverse myelitis. The first case to be reported is one of complete transverse myelitis.

The patient was a primipara of twenty years, white, of good heredity, the past history irrelevant, with no illness save the usual diseases of childhood and with no history of trauma. The girl became pregnant a few months after marriage, the pregnancy proceeding normally for the first three months. She then noted some weakness of the legs together with occasional shooting pains. The weakness became progressively worse and extended from the legs to the thighs.

She was seen by one of us in consultation with Dr. George E. Shaffer, when seven months pregnant.

At this time there was complete motor paralysis of both legs; the skin was anesthetic and did not react to a pin prick although there had previously been some hyperesthesia noted. The patellar reflexes were absent and the muscles of the legs and thighs were flabby and presented no contracture or spasticity.

The bladder was completely paralyzed, a period of retention having been followed by urinary incontinence. The sigmoid and rectum were paralyzed, the manual removal of feces being obligatory. The chest was negative, and the reflexes above the level of the first lumbar vertebræ were unchanged.

The blood showed a moderate anemia with a leukocytosis of 10,000. Wassermann reactions of both husband and wife were persistently negative. In view of the conditions found a diagnosis was made of complete transverse myelitis and hospitalization and termination of pregnancy by cesarean section was advised. Both were refused and the patient became steadily weaker, more toxic as a result of kidney and bowel retention and finally died of toxemia and exhaustion when ten months pregnant, the fetal heart having ceased to beat some two weeks before the death of the mother.

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*Read at a meeting of the Philadelphia Obstetrical Society, February 2, 1922.

There was no uterine contraction nor any evidence of beginning labor at any time. Autopsy was not permitted.

Several analogous cases are noted in a cursory survey of the literature but it is noteworthy that in analyzing the labors of patients suffering from transverse myelitis it is found that most of these develop painless uterine contractions and in general are delivered spontaneously, practically without the use of the abdominal muscles.

Whether this is due to the location of the spinal lesions, the cutting off of the fibers at a higher level in one case than in the others is not known, but as the uterus is under control of the sympathetic, we cannot consider this a factor.

Two suggestive case reports are appended.

Robinson, G. D. Proc. Royal Soc. Med. (Sec. Obst. and Gyn.) 12:22. Jan., 1919, reports a case of labor in a paraplegic woman. The patient, aged thirty-one, was a seven para with one miscarriage. Attack began with four days' aching pains followed by paralysis of legs and abdominal muscles, with retention of urine and feces. Bedsore developed. Leg reflexes absent. Pain and tenderness in back at about tenth dorsal spine. Later there was incontinence of urine and feces. Diagnosis: Complete transverse myelitis in the dorsal region. At term this patient delivered herself spontaneously of a living female child. Labor was painless and unassisted by the abdominal muscles. Involution proceeded normally and the patient made an uneventful though slow recovery.

Meyer C., Zentralbl. f. Gynäk., 44:238, March 6, 1920. Reports a case of a spinal cord tumor in pregnancy. A twelve para, three months pregnant, seemed to be suffering from multiple sclerosis. Following some treatment for the condition she began to have severe pain and twitching in legs with the development of a flaccid paralysis in the right and spasticity in the left. For two months there was incontinence of urine and feces. K. J. reflexes were present in both sides, left stronger than right. Babinski, left doubtful, right strongly positive. Speech and sight unimpaired.

At term patient went into painless labor and was delivered of a fetus with occiput posterior, without use of abdominal muscles and without sensation other than a slight pulling.

After delivery a decubitus ulcer developed and the patient subsequently died.

Autopsy disclosed a diffuse intramedullary tumor in lower cervical and upper thoracic region, which was found to be a sarcoma.

A consideration of these cases inevitably leads to the query as to what association exists between pregnancy and acute inflammatory diseases of the cord. According to Coplin the etiology of acute myelitis is probably always infective, toxemic, traumatic, hemorrhagic, or embolic.

Inasmuch as the toxemias of pregnancy are still almost unexplored territory to the pathologists it may well be that some bizarre trend of the toxins originating during gestation affects the cord. This relation is at best, however, a remote one and the writers fail to find

in the occurrence of myelitis in pregnancy anything more than a coincident infection.

The treatment of this complication of pregnancy is unsettled, there being no definite rule established presumably by reason of the rarity of the associated conditions. In the three cases quoted two of the patients delivered themselves spontaneously and apparently without the use of the paralyzed abdominal muscles. In the third case there was no attempt at labor, no uterine contractions, the child dying postmature and the mother eventually succumbing without any evidence of labor. Two of these cases resulted fatally, one recovering. It would seem that in view of the very grave nature of transverse myelitis, the child should be sacrificed in the interests of the mother whenever the cord lesion is apparent before the time of viability. If the condition should be due to a pregnancy toxemia, certainly the source of such toxemia must be eliminated and if, as is probably the case, the myelitis is purely coincident, the mother should be saved from the strain of pregnancy that all of her bodily resources may be utilized to combat the dangerous infection from which she is in peril of her life.

Therefore we think pregnancy should be interrupted in the presence of acute transverse myelitis if the lesion is discovered before the viability of the child.

When the child is viable, the patient should be delivered as soon as practicable with a view towards saving her from any longer pregnancy than is absolutely necessary for the life of the infant. The method of delivery will vary with the individual case, from induction of labor by bougies or bags in the less advanced case to cesarean section preferably under local anesthesia in the grave cases. Delivery should be performed in some manner at the earliest possible moment. The puerperium of victims of myelitis who survive labor, should be managed with regard to the general systemic treatment of the disease and especial care must be taken to prevent the development of decubitus ulcers to which such patients are so prone.

Turning now to multiple neuritis the writers desire to report the following case:

A para ii of twenty-six entered the Frankford Hospital with a history of one instrumental labor with a dead infant.

The present pregnancy had been noteworthy by reason of protracted hyperemesis during the first four months with persistent indigestion and evidences of toxemia recurring all through the pregnancy. The woman entered the hospital in labor the membranes having ruptured some time prior to admission. On examination she was found to be a fairly well nourished woman, the chest negative, the abdomen much enlarged by the presence of a large fetus approximately 52 cm. in length. The pelvis showed a moderate anteroposterior contraction with true conjugate of somewhat under 9 cm. In view of the previous obstetric history cesarean section was advised and accepted; the operation proceeded without incident, a high

incision Sänger section being done. Mother and child progressed uneventfully for six days when the mother suddenly complained of intense pain in the left arm and shoulder. Embolism was feared but the condition remained unchanged for two days, the only evidence of illness being a slight elevation of temperature and the intense pain. Suddenly the right foot and lower leg became the seat of severe pain while the shoulder and arm improved. At this time the patient developed a train of mental symptoms with fits of loss of memory and marked apathy alternating with restlessness and uncorrelated delusions. With this development it became apparent that the case was one of muliple neuritis with toxemic cerebropathy or as it is generally termed the Korsakoff syndrome, which was first described by Korsakoff in 1887. At this time the temperature was slightly febrile, there was a moderate leukocytosis and the urine revealed a trace of albumen. There was a considerable loss of power in the extremities affected by the neuritis, which power was regained when the pain in that particular extremity lessened, the time averaging about forty-eight hours. The symptoms were to a considerable degree controlled by the exhibition of salicylates and after four weeks gradual improvement took place. At the end of two months the patient was well.

The association of multiple neuritis and pregnancy is a fairly common one, cases being not infrequent in all large maternity clinics. The disease frequently manifests itself during the latter half of pregnancy, when it is termed neuritis gravidarum. This variety of inflammation is usually not so severe or so persistent as that developing after delivery and known as puerperal peripheral neuritis.

These cases are so common and it is so usual to find neuritis occurring in a patient who presents definite signs of toxemia in some form or other, that a connection between the two seems obvious; but the pathogenesis is obscure and other than ascribe the neuritis to some indirect reaction to toxins, no mechanism for its production can be described. The association of the Korsakoff syndrome is not so common but mild cases of impaired memory and transient dementia do occur more frequently than is thought, the cerebral symptoms being so mild as to escape detection.

The attacks of neuritis come on suddenly, any extremity or group of extremities being affected, and the pain is apt to suddenly shift from one group of peripheral nerves to another. Slight elevation of temperature is the rule and there is ordinarily some leukocytosis.

The treatment is to be directed toward the elimination of toxins by general measures and active steps must be taken to relieve the severe pain. Splinting the affected extremity, counterirritation and the exhibition of salicylates internally seem to give the best results. Opiates are commonly required during the first stages of the disease.

124 South Eighteenth Street.
1225 Spruce Street.

(*For discussion, see page* 91)

ABDOMINAL PREGNANCY AS OBSERVED IN THE CHARITY HOSPITAL OF NEW ORLEANS

By P. Graffagnino, M.D., New Orleans, La.

(*From the Department of Gynecology, Tulane University.*)

DURING the year 1920 the writer, in reviewing the cases of extrauterine pregnancy recorded at the Charity Hospital, New Orleans, from 1906 to 1920, found so many interesting cases of advanced extrauterine, or so-called abdominal, pregnancies, that he deemed it of some scientific value to record these cases, an added incentive being given by the fact that it was his privilege recently to have such a case come under his own immediate observation.

Alfred C. Beck, in the *Journal of the American Medical Association*, September, 1919, and John M. Maury, in *Surgery, Gynecology and Obstetrics*, November, 1920, have so thoroughly covered this subject that anyone interested in it should by all means read their reviews and their most valuable conclusions.

In this paper I will record only those cases in which the diagnosis of advanced abdominal pregnancy was positively established by operation, x-ray, or the passing of fetal bones. From 1906 to 1920 approximately 18,835 cases were admitted to the various gynecologic services of the Hospital, and among the cases recorded as extrauterine pregnancy, only eleven cases could be considered as positively filling the above specifications. The first case is the writer's own, the others are summarized from the Hospital records.

Case 1. Colored female, age twenty. Occupation: housework. Previous history negative, menses regular, with severe dysmenorrhea. Married at 13, one child eight years old, normal delivery. No miscarriages. She had missed her periods for eight months, then they reappeared three months prior to her admission. When they reappeared, she states that she passed some sort of sac, and she felt life until this resumption of her periods. About ten days before admission her local doctor drained off a gallon of bloody fluid by inserting a trocar in the midline between the umbilicus and pubis. She had been lactating for three months. Bowel function regular, some nocturia. Phyical examination showed the abdomen to be that of full term pregnancy, with parts palpable, but no heart sounds heard. Chadwick's sign present. The cervix was small and anterior lip was slightly softened, but not enough so for an advanced pregnancy. Behind the pubis was a hard mass about the size of a fist, apparently connected with the cervix, and perhaps the fundus of the uterus. Above the mass in the abdominal cavity was a large, irregular, hard mass, about the size of a seven months' pregnancy. The x-ray report was that a fetus was present within the abdomen, with the head to the right; other laboratory reports were negative. Four days after admission, under ether,

a median incision was made, extending about two inches above the umbilicus. Within the cavity was found a large sac containing a full term, macerated fetus, the position being almost transverse, with the head in the right iliac fossa. The fetus was delivered without difficulty, but the placenta was intimately adherent to the fundus, and almost to the posterior surface of the uterus. It appeared to have undergone a waxy degeneration, and it was almost impossible to separate it from the uterus, so subtotal hysterectomy was done, taking off the fundus above the level of the tubes and ovaries. The sac, which had also undergone waxy degeneration, was so intimately adherent to the parietal peritoneum and the abdominal viscera, that most of it was allowed to remain *in situ;* what could be peeled off without injury to the surrounding structures was removed. Closure without drainage. The patient made an excellent recovery, the wound healing by first intention, and was discharged thirteen days after operation.

The fetus weighed 2812.32 gm., and was 45.72 cm. long. The F. O. diameter was 11.0 cm., B.P., 9.0 cm. The placenta weighed 952.56 gm., was 12.5 cm. in diameter, and 6.0 cm. thick. The cord was 35.56 cm. long.

CASE 2. August, 1920. Colored female, aged thirty-one. Previous history negative. She stated that she considered herself eight and a half months pregnant, but could not recall the date of her last menses. At no time had she had any feeling of faintness, sharp pain, or any symptom which would suggest tubal pregnancy. Forty-eight hours before admission she considered herself in labor, though the pains were cramping rather than bearing down. She was in bad condition when admitted, her pulse weak and rapid and she gave signs of exhaustion. The examination was negative, except for a hard mass at the left of the umbilicus, taken to be the fetal head. Operation the day after admission, under ether, in the midline. A full term, viable fetus, which lived only a few hours was delivered, but the placenta was very firmly adherent and was not removed because of the fear of hemorrhage. Closure with abdominal drainage. Following operation she was very restless, with pain, and nausea not relieved even by gastric lavage. Her temperature ranged from 102° to 104°. On the sixth day she had a profuse hemorrhage through the wound, following removal of the drain. She was put on the table at once, almost cyanosed, and with a very poor pulse, and the placenta and membranes removed under nitrous oxide. Saline infusion was given on the table, but her pulse was imperceptible at the conclusion of the operation and she died five hours later.

CASE 3. This was a colored female, age ninety, who entered the Hospital Sept. 28, 1920, complaining of shortness of breath. The previous history was negative, menstrual history without incident. She had had no children, and had had her menopause at forty. It would be well to remark that the patient was of a low grade mentality, and her history was secured with difficulty. The physical examination revealed a hard mass fixed in the pelvis at the left, suggesting a fibroid, the other findings being irrelevant. Fourteen days after admission the patient died, the diagnosis after autopsy being cardiac hypertrophy and dilatation, acute nephritis, and chronic passive congestion of the liver and spleen. The peritoneal surface was smooth, moist and glistening, with a small amount of free fluid throughout. The uterus, which presented apparently a leiomyoma undergoing calcification, extended above the brim of the pelvis, and when opened, was found to be posterior to this mass described as a leiomyoma. The right tube and ovary were easily traced out, the left tube was obliterated and apparently surrounding

the mass previously described. The left ovary was small. Sagittal section of the mass revealed a calcified, well defined fetus.

CASE 4. September, 1920. Colored female, age thirty-four. Occupation, housework. Previous history negative. Menstrual history regular. According to her statement, her menses were last seen eighteen days before admission, though her present trouble had begun ten months previously, with nausea, vomiting, and severe pain in the left abdomen, which continued intermittently for two months. Two months later the pain continued in the right iliac and inguinal regions, and was stationary. About this time a mass appeared in the abdomen, which increased in size, and was slightly movable. The woman considered herself pregnant. Examination was negative except for a large mass like a fibroid in the region of the liver. Vaginally, the cervix was hard, and a mass was palpable at the left, which seemed to be the uterus.

Under ether, an exploratory laparotomy was done in the median line, and a mummified seven months' fetus was found free in the cavity. The placenta was attached to the body of the uterus and to the right tube. The fetus was removed, and supravaginal hysterectomy was done, with right salpingooophorectomy. The patient had gall stones, but nothing was done in that region. She was discharged as cured sixteen days after operation.

CASE 5. November, 1917. Colored female, aged twenty-nine. Previous history negative. Menstrual history without incident. Seven children, the last being stillborn. At the time of admission she considered herself almost nine months' pregnant. Three months after the onset she saw blood for two days, otherwise she had a normal course. Five days before admission she began to have severe abdominal pains, which she insisted were not labor pains, and from that time she saw blood. The physical examination was negative, except that the abdomen was pendulous. Fetal parts were palpable on the left, and the breech extended three fingers above the umbilicus. On the right was a large, soft mass, apparently independent from the fetus. Vaginally, the head was well engaged in the pelvis, in the right oblique diameter, with the face under the pubis. Over the head was a thick membrane, apparently the posterior vaginal vault, and no cervical ring could be made out. The cervix could be felt high up behind the pubis, pushed to the right, and connected with a mass on the right of the abdomen, which was taken to be the pubis. Laparotomy was done under ether in the midline, and a dead, full-term fetus, weighing 5.88 pounds, was extracted, left salpingooophorectomy being done also. The amniotic sac was plastered over with omentum, which stripped off easily, and there were no adhesions to the intestines or abdominal wall. The uterus was large and boggy and pushed to the right, and about the size of a three months' pregnancy. The placenta was attached to the old ruptured tube, the ovary, and the upper portion of the broad ligament, and nourished by the ovarian artery and uterine anastomosis. The ovarian artery was hypertrophied to the size of the internal iliac. The patient was discharged as cured seventeen days after operation.

CASE 6, October, 1916. Colored female, age thirty-eight. Previous history negative, except for an attack of acute P. I. D. (?) two years previous, treated by douches and rest in bed. Menses regular, last seen eight months before. She had had leucorrhea all her life, but none for the last seven months. She had first felt life five months before admission, but had felt none for the last two weeks. For the last two months she had had unusual abdominal swelling, with a feeling of

tightness, pain in the right lower abdomen and lower back, and sensations of dizziness. She was poorly developed and nourished, heart rapid but regular, milk in the breasts, and the abdomen was so distended that the diaphragm was pushed upward. The walls were very tense and fluid seemed to be present. Vaginally, the cervix was almost obliterated and pushed to the right. There was a small mass at the left, probably the displaced uterus, or an ovary. No ballottement. Six days after admission laparotomy was done, under ether, in the midline, disclosing a large sac adherent to the abdominal wall anteriorly. It was partially freed and aspirated, revealing a blood-stained, brownish fluid, with yellow flocculi and shreds of tissue. The fluid was withdrawn by trocar. When the sac was opened, a full-term dead fetus was delivered, and the cord clamped. As the placenta was firmly adherent, it was not separated from the sac wall, but the sac was seized between clamps and freed from the entire abdominal wall, omentum, intestines and mesentery. The omentum was ligated *en masse* and the sac delivered. In the pelvis were found two small fibroids to the left of the mass which was taken to be the uterus, at the right of which the sac arose. The cervix presumably led into this mass, as well as into the uterine cavity. The left tube and ovary were intact, the right could not be differentiated. The sac, the uterus, and the left tube were removed *en masse*, and later the cervix was removed, and a pack put into the vagina leading to the pelvis. The abdomen was also drained through a stab wound in the left.

The patient made a good recovery and was discharged as cured twenty-eight days after operation.

CASE 7. August, 1913. Colored female, age thirty-four, occupation, housework. The only statement secured from this woman, as she deserted almost at once, was that for some time past she had been passing by rectum what she took to be fetal bones. An x-ray was taken, which showed bones, presumably the femur, in a sac adherent posteriorly, and communicating with the sigmoid. Then, as stated, the patient deserted, and her subsequent history could not be secured.

CASE 8, July, 1910. Colored female, age twenty-six. Previous history negative. Three children, one living, one miscarriage. Ten months before admission she was taken with sudden abdominal pains and was in bed for three months. She saw no menses for five or six months, then her periods were regular again, though more profuse. Three months after this attack, and ten months before admission, she felt fetal movements and considered herself normally pregnant. Throughout this period she had had severe abdominal pains and, towards the last, there was unusual swelling and tightness of the abdomen. Examination showed the abdomen to be markedly distended, with the walls very tense and sensitive to pressure. Vaginally, the cervix was almost obliterated and pushed to the right side of the vault, and a small mass to the left was taken to be the uterus. Laparotomy was done, under ether, in the midline, and a large fetal sac, containing a dead fetus, apparently full term, was found in the region of the left tube. The dense adhesions, particularly of the bowel, were dissected, and the tube cut close to the left cornu. A cyst of the broad ligament on the right was also removed. The patient made a good recovery, and was discharged as cured seventeen days after operation.

CASE 9, April, 1909. Colored female, age twenty-eight. Previous history negative. Menses regular. One living child, one miscarriage. At the time of admission she had not menstruated for 18 months. Four or five months after their cessation she had been taken ill with nausea, vomiting, and severe abdominal

pains, and about five months after this attack she had seen bones projecting through the abdominal wall in the region of the umbilicus. She came to the Hospital with a bottle of fetal bones, and the presumption was that she had removed them herself. Physical examination showed a large ulcer near the umbilicus, which presented a foul discharge, and from which a tiny bone was then projecting. Under ether this ulcerated sac was opened, without entering the peritoneal cavity, and the remains of the skeleton of what appeared to be a four months fetus were removed. There was a communicating passage between this sac and the lower bowel, which later closed spontaneously. She was discharged thirteen days after operation, but returned to the clinics for treatment until the superficial sinus was completely closed.

CASE 10. April, 1907. Colored female, age twenty-eight, occupation, housework. This woman was admitted with a very vague history of some large mass in the lower abdomen, associated with high temperature. Operation was advised by her local physician, who does not seem to have considered her pregnant. Vaginal examination, under ether, revealed a condition resembling pelvic abscess, but when an attempt was made to puncture the mass, the sponge holder was withdrawn with a fetal arm. Immediate laparotomy was done in the median line, and two dead feti of different ages were removed. The history is most disappointing, giving absolutely no further data concerning them. The patient herself, after a stormy convalescence, was discharged as cured on the sixty-first day after operation.

CASE 11, November, 1906. Colored female, age 26, occupation housework. Nothing of note in her previous history, menses always regular. She was admitted with a diagnosis of probably dead fetus. The external conjugate was 19 cm., the intercristal 25 and the interspinous 24. Vaginally the cervix was found to be hard and elongated, hypertrophied and not patulous. A mucopurulent discharge was present. Sixteen days after admission laparotomy was done, under ether, in the median line, and a dead, full term fetus was found, covered with membranes that were adherent to the bowels, uterus and appendages. The fetus was first removed, and then a large mass, evidently the placenta, with a large, organized clot of blood, which had become encysted in the fimbriated extremity of the left tube, was ligated and removed *en masse*. The adherent membranes were removed piece by piece, and because of the dense adhesions, right oophorectomy with partial right salpingectomy was also done. The patient made a good recovery and was discharged as cured twenty-eight days after operation.

Summarizing these eleven cases, it is interesting to note that in every instance the patient was a colored woman. The ages range from 20 to 90, in the last instance the diagnosis being established by post-mortem many years after the pregnancy occurred. It is also interesting to note that the only fatality in the series of operated cases occurred in the only case where the fetus was viable. In the other cases, where the operation was deferred beyond term, or where the child was taken before the patient was at term, the results were uniformly successful, proving that abdominal section, where the diagnosis of abdominal pregnancy is established, has no higher mortality than ordinary gynecologic surgery, provided the operation is timed correctly.

Society Transactions

THE NEW YORK OBSTETRICAL SOCIETY

MEETING OF FEBRUARY 14, 1922

THE PRESIDENT, DR. R. H. POMEROY, IN THE CHAIR.

Program Contributed by the Members of the Staff of the Sloane Hospital

DR. WILLIAM E. CALDWELL demonstrated a **New Leg-Holder for Gynecologic and Obstetric Use.**

The leg-holders here illustrated (Fig. 1) are designed to support a woman's thighs and legs during obstetric and gynecologic operations, without putting strain

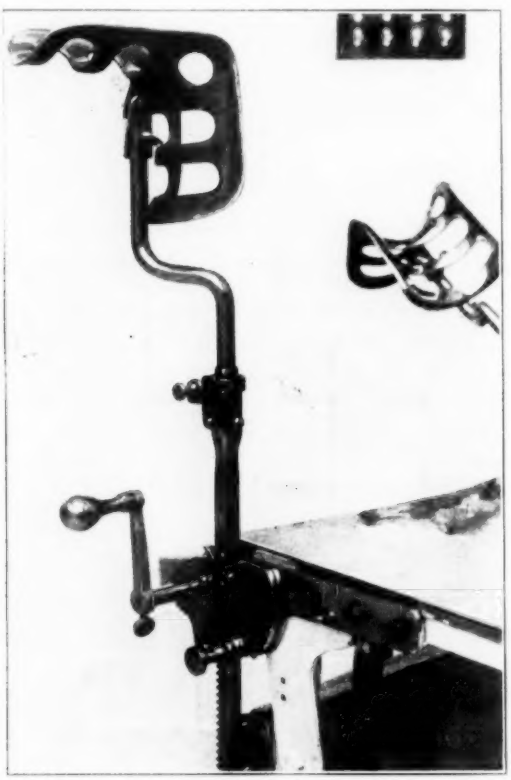

Fig. 1.

on the lumbar or abdominal muscles or pelvic joints, and without distorting the soft parts of the genital tract. They also allow easy changing from one position to another, frequently necessary in obstetrical and gynecologic operations, without

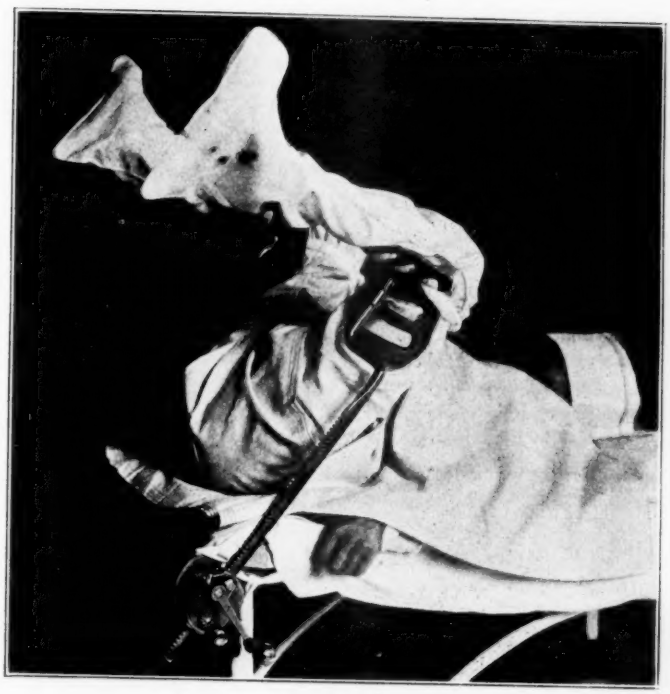

Fig. 2.

Fig. 3.

disturbing the surgical "set-up." These leg-holders allow combined vaginal and abdominal manipulations or permit the placing of an assistant between the thighs in gynecologic operations, as is practiced in Wertheim's clinic. They are easily adjustable from the side, and the pelvis can be tilted readily into various positions.

The crutch proper is a bronze casting, shaped to hold the knee comfortably. It is on a swivel joint, permitting easy, comfortable adjustment and is held securely in position by tightening the thumb screw. The crutch is connected to a round, cold-rolled steel rod, terminating at a swivel joint which permits adjustment of the crutch from right to left, thereby increasing or decreasing the distance between the two crutches. A spring catch, operating in grooves, holds the crutch firmly in place, after the desired position is obtained. To the joint is attached a milled, cold-rolled steel square rod, which operates in the raising and lowering mechanism. This mechanism consists of a set of milled steel gears concealed in a metal casing. The gears are turned by a removable swivel hand crank. A ratchet stop holds the crutch firmly in place when set at the desired height. The crutch may be swung to any desired position from the horizontal to the vertical, and then set by means of an additional strong spring catch, operating in a series of bores and firmly held in place when set. By this adjustment the crutch may also be swung completely around and down to the side, so as to be out of the way when not in use. The crutch is attached to a bracket equipped with bores and bolts so that it may be attached to any operating table.

These leg-holders allow us to place a patient in position with thighs markedly extended and to raise the thighs by means of the swivel crank all the way back to extreme flexion (Fig. 2), or to bring the thighs from extreme flexion downward into a position of extreme extension (Fig. 3), the rods shortening as the thighs are brought down and lengthening as the thighs are brought up. Thus, any degree of extension or flexion can be obtained and the position held by means of the large pin next to the crank.*

DISCUSSION

DR. ROBERT L. DICKINSON.—The textbooks on gynecology though largely operative, may be said to give no consideration to the organization of the operating room, and of the operation as such,—even the books on operative gynecology, save Doederlein, being very incomplete. No gynecology considers whether a leg-holder may give backache or damage a sacroiliac joint, nor do they advise the moderate flexion of the thighs in a laparotomy which will relax abdominal muscles, nor a little pillow under the lumbar spine at operation to save grievous postoperative backache. My association with an orthopedist has demonstrated the unnecessary damage to sacroiliac joints. In women with long legs, with relaxed muscles, or with sacroiliac joints predisposed to injury, the ordinary method of hanging the legs by the ankles is reckless disregard of joint injury. Postoperative backache is added distress which our patients may well be spared. Thrombophlebitis is favored by any of the crutches that press in the popliteal spaces, and probably by an unpadded Goepel knee trough. The ankle-hung foot is a device especially planned to put the foot in the way of each assistant. He must duck under it or stoop over it. The thigh bent on the abdomen and the leg on the thigh gets the lower limb more effectively out of the way than any other scheme, but it, too, inflicts damage on the pelvic joints and adds kneeache thereto, relaxed as everything is under anes-

*The Sloane Hospital is indebted to Miss Marie Louise Duchesne for working up many of the details of the leg-holder with the Hospital Supply Company of New York.

thesia. To merely let the assistant shove the leg aside produces the same result. The leg trough of Goepel renders the most complete support and has this advantage, it is useful in both abdominal and vaginal work. For laparotomy it gives the slightly bent thigh that relaxes the abdominal muscles and holds the patient from slipping (the shoulders of course, taking most of the weight), while the patient can be quickly swung into position for vaginal work, the legs held by the same device, and without change in the leg drapes.

The support of this device presented by Dr. Caldwell is better than the Goepel universal joint, in that the joint is difficult to keep locked, and because the new arm permits elongation as the Goepel stalk does not. A sleeve on the Sloane device would allow the legs to swing open more widely and freely. If Dr. Caldwell's holder could swing far down enough to give the Walcher posture we should have every possible demand satisfied.

Dr. Warren Hildreth presented a report of a case of **Premature Separation of a Normally Implanted Placenta.**

The patient was 34 years old. She had never had scarlet fever, diphtheria, tonsillitis, venereal disease or operations. The only fact bearing on her former kidney condition is that she was refused for life insurance in 1915, on account of an unsatisfactory urine specimen. Her menstruation began at 18, every 28 days, normal in duration and amount, and without pain. Her last menstrual period was August 15, 1919, making her probable date of confinement May 22, 1920. During her pregnancy, she was never ill. She saw a doctor only once during her antepartum period. From May 1st, about three weeks before term, she first noticed swelling of the ankles. However, she continued her work as a dressmaker, fitting and draping until 7:30 each evening. She laced her corsets almost as tightly as before her pregnancy, so that none of her friends knew of her condition. She, frequently, had considerable abdominal pain after bending, while tightly laced.

On May 6, two weeks before term, she worked particularly hard, went to bed and slept well until 4 A. M. of May 7, when she awoke with severe abdominal cramps and moderate vaginal bleeding. She was seen by a physician at about 10 A. M. and sent to the hospital. On admission, her general condition was excellent, her pulse was 90, her systolic blood pressure 150, and her urine contained a heavy trace of albumen. On examination of her abdomen, the uterus was found to extend to a point 3 cm. below the ensiform. It was hard and fairly contracted, but relaxed slightly at times. It was impossible to make out the fetus, and there was no fetal heart and no ballottement. On vaginal examination, the head was found presenting and the cervix 1 cm. dilated, soft, and about 2 cm. thick. No placenta was felt.

Without anesthesia, a No. 2 Voorhees bag was inserted, hoping to deliver patient from below. After the insertion of the bag, the patient rested quietly and slept most of the following night. When examined on the morning of May 8, the bag was found still tightly wedged in the cervix. The uterus was still regular in outline, but now extended to the ensiform. It was board-like and had no periods of relaxation. Although the patient had a normal temperature and a pulse of 80, she looked anxious and did not seem to be in as good condition as one would judge from her pulse and temperature. On consultation with Dr. Studdiford, and under his direction, we did a cesarean section. Under ether anesthesia, the abdomen was opened with a low median incision. There was a large amount of blood-tinged

fluid in the abdominal cavity. The uterus was very slightly rotated to the right. There were huge, deep purple veins making a large mass either side of the uterus. The uterus, tubes, ovaries and broad ligaments were swollen, edematous, and almost black in color. There were numerous large ecchymotic spots scattered over the surface of the uterus, directly under the peritoneum. The uterus was opened and the placenta found completely separated, with about a quart of old clotted blood between the placenta and the uterine wall. The placenta had been attached to the anterior portion of the upper uterine segment. The placenta was a deep purple color, and its surface was covered with old clots. The child was 50 cm. long and weighed 7 lb. 4 oz., and had apparently been dead for some time.

After emptying the uterine cavity, the wall of the uterus was observed for a number of minutes. The muscle remained flabby and showed no tendency to contract, except over a small portion of the lower segment. There were large thrombi in the veins along the cut edge and the color of the uterus changed very little after the relief of the tension. Therefore, a supravaginal hysterectomy was done, fearing to leave the uterus on account of later necrosis and infection, or postpartum hemorrhage from its atonic walls. Along with the uterus the tubes and ovaries were removed, because of their swelling and engorgement.

Pathologic Report, by Dr. W. C. Johnson.—In the hemorrhagic areas, the decidua is normal in thickness. It shows no hemorrhage or necrosis, but is rather diffusely infiltrated with a considerable number of polymorphonuclear leucocytes. The myometrium shows pathological alterations throughout its entire thickness. There is a moderate edema of the interstitial fibrous tissue, separating the bundles of smooth muscle. Throughout these edematous areas, around blood vessels, also frequently invading the bundles of smooth muscle, is a variable amount of cellular exudate.

There are no hemorrhages in or adjacent to the decidua, but passing outward toward the peritoneum, at first small hemorrhages are found between muscle bundles, then the hemorrhages become larger and, finally in the outer quarter or fifth of the uterine wall, the hemorrhages are so large and confluent that the muscle fibers appear as small islands entirely surrounded by blood. The smooth muscle fibers show the hypertrophy characteristic of pregnancy, but do not appear as sharply outlined and distinct as in a normal contracted postpartum uterus. The fibers in the hemorrhagic regions, especially in the groups surrounded by hemorrhage, are pale and swollen. In spite of the diminished number of muscle nuclei visible, and the altered appearance of the muscle cells, it is not possible to state definitely that any of the cells are actually necrotic.

The blood vessels through the uterine wall do not show any significant changes; no vascular lesions can be found which would explain the hemorrhages. Sections of the uterine walls show no organisms. The placenta shows no changes of any significance.

There are three conditions present: 1, inflammatory reaction; 2, interstitial hemorrhage; and 3, degeneration and possible necrosis of muscle fibers.

Any one of the three may be primary. It is possible that the inflammatory exudate is not directly related to the other lesions, but is simply a reaction to the separation of the placenta and the irritation of the retained placenta and blood clot lying within the cavity of the uterus. This seems the simplest explanation, as the reaction is greater toward the decidua, while the hemorrhage and muscle changes are more marked toward the peritoneal surface. This still leaves unsettled the relation between the hemorrhages and the degenerated changes in the muscle fibers. The muscle cells had no alterations sufficiently marked to lead one to be-

lieve that an adequate explanation of the condition could be based on muscle degeneration and weakening.

On the other hand, the condition of the blood vessels does not indicate either venous stasis or lesions of the vessel walls which might lead to rupture. Two changes occurred, either degeneration of muscle fibers, or interstitial hemorrhages of the myometrium.

The patient's postoperative course was uneventful. The albumen, which only once showed as a sediment on boiling, quickly cleared up and her systolic blood pressure fell from 150, just before operation, to 110 on discharge. Since operation, the patient has had no symptoms referable to the pelvis. She has had no hot flashes and is no more nervous than before operation. Her systolic blood pressure now is 130; her urine contains no albumen or casts.

We report this case to demonstrate an excellent result following cesarean section and hysterectomy. Among the cases classed as accidental hemorrhage, there are a certain number which show large subperitoneal hemorrhages, as well as large hemorrhages into the outer portion of the myometrium. In these cases, the shock is usually considerable. The uterus is too severely injured to react to any stimulus tending to delivery from below. For this reason, and because of the subsequent danger from infection and postpartum hemorrhage, the delivery should be by cesarean. The question of hysterectomy in each case would depend on the condition of the uterus at operation. It seems that the mortality would be greatly reduced by following this less conservative treatment.

Dr. Willson, in the January, 1922, number of *Surgery, Gynecology, and Obstetrics*, reviews the literature and gives a maternal mortality of 55 per cent for similar cases. In this case, there were present two causes for the hemorrhage: 1, toxemia, and 2, venous stasis from extremely tight lacing, a fact of great importance according to Morse.

DISCUSSION

DR. A. H. MORSE, NEW HAVEN, CONN.—This very interesting case illustrates the most serious type of separation of the normally implanted placenta with concealed hemorrhage. The dissociation of the myometrium of which Dr. Hildreth spoke is the type of lesion which we have found in all the cases which we have had in New Haven. In going over the sections from the uteri of these cases I was unable to demonstrate in the vessel walls the changes which Dr. Williams mentions in his paper On the other hand in one case in which the uterus failed to contract following delivery, and in which hemorrhage made a supravaginal hysterectomy necessary, there was thrombosis of the veins of the broad ligament. Sections taken through the lateral wall of the uterus and the broad ligament showed definite laminated thrombi; in other words it was clear that in this case thrombosis occurred before operation.

Now it happens that last night we had a case of premature separation of the placenta of the less serious type. The patient entered the Hospital in the eighth month of pregnancy with a systolic pressure of 155 and with slight uterine bleeding. Several years previously she had suffered from a nephritic toxemia, with convulsions. Since the uterus was contracting upon admission, we felt that in spite of a partial separation of the placenta no radical treatment was indicated. However, the cervix was slow in dilating, and as the bleeding became a little more profuse, we inserted a Voorhees bag. This morning she was delivered of a stillborn child. The uterus contracted satisfactorily and there was no free bleeding during the third stage. The placenta showed a large collection of blood clots with

a flattening of the placental tissue beneath it. There was probably no hemorrhage into the myometrium in this case.

It is interesting that there had been tight lacing in Dr. Hildreth's case. This perhaps caused a disturbance of the venous circulation. I do not believe that a marked rotation of the uterus is necessary to bring about such a disturbance. Moreover the position of the uterus as we see it when the abdomen is opened may differ from the position which the organ occupies previous to operation.

I have discussed the question of toxemia as a cause of this complication of pregnancy with biochemists, and we have difficulty in conceiving of a toxin which would so acutely affect the blood vessels of the uterus and no others. Of course the question is not entirely settled but I believe that it is due to an interference with the venous circulation.

DR. C. PAUL HUMPSTONE.—Whatever may be the cause of this particular condition which is associated with toxemia and hemorrhage into the uterine muscle and ablation of the placenta, in dealing with it we have come to recognize that there must be a different treatment applied to the cases which are in labor with some dilatation, and the cases which are not in labor.

I have handled seven cases of ablation of the placenta, in which we did cesarean sections, and found that there was no necessity to take out the uterus in any one of these cases, and not a single one of them died. While the uterus seemed soft and flabby and had this bluish-purplish appearance underneath the surface, nevertheless we sewed all these uteri up, and in due time they contracted without undue hemorrhage from the vagina and got well.

We have at the present time a case in the hospital that came in with 3½ fingers' dilatation, with a dead baby. She was 7½ months pregnant, with ablatio placenta. We simply incised the cervix and pulled the baby out with the forceps. She had no unusual hemorrhage after packing the vagina.

Are we not unduly alarmed about postpartum hemorrhage in these cases? We don't find many fatal cases reported. My experience has been that by simply doing a section and sewing them up, they get well and may have another baby.

DR. JOHN O. POLAK.—It is our custom, in these cases, when admitted, to measure the fundus and mark the size of the uterus, and the blood count and hemoglobin are taken at very frequent intervals, as well as the pulse, and if abdominal distention is accompanied by a rise in pulse and particularly a drop in hemoglobin, which is very rapid in these cases, a hysterectomy is done. Most of these cases, however, can be treated on the expectant plan.

DR. J. MILTON MABBOTT.—The case raises the question as to when involution begins. Does it begin when the uterus is emptied, or when the child dies, or when the placenta is completely separated from the placental site in the uterine wall?

In this case we can assume that the placenta was fully separated about three days before delivery. If we assume that involution began three days before delivery, the fatty degeneration of the muscular fibers, due to involution, would have progressed so far that that would account for the failure of the uterus to respond to the dilator. In other words, the failure of the uterus to contract and expel its contents, would naturally make the cesarean operation a proper means of emptying the uterus under those conditions.

Dr. W. W. Herrick read a paper (by invitation) entitled **Pregnancy and Heart Disease from a Medical Viewpoint.** (For original article see page 1.)

DISCUSSION

DR. GEORGE N. SLATTERY (by invitation).—Dr. Herrick's quotation of Dr. Mackenzie should be a matter of particular authority, both from the standpoint of the cardiologist and the obstetrician. It will interest you to know that he, while bearing international fame as an authority on the heart, had an experience with many laparotomies and obstetrical cases. He was a general practitioner in the broadest sense. This would seem to qualify him particularly as an authority on such a subject as pregnancy complicating cardiac diseases. His big contribution was the investigation of heart cases from the standpoint of disturbed rhythm. In a practical way he stressed the importance of remote signs rather than the immediate heart murmur. I wish further to quote him as to the importance of signs rather than murmurs. By way of example, pectoral tenderness and anginoid pain may be of more grave prognosis than the usual signs of broken compensation.

I feel somewhat qualified to endorse Dr. Herrick's comment concerning anesthesia. In a year or two, an opportunity was given me of observing the circulation from the anesthetist's viewpoint. This experience emphasizes two points brought out by Dr. Herrick, namely: first, the importance of relief from apprehension during the pre-anesthesia stage; second, that ether is definitely the anesthetic of choice in cases where the circulation is a particular consideration.

I have come in contact from time to time with obstetricians who have asked my opinion as to whether certain heart conditions would permit the continuance of pregnancy. These cases often were considered by the men sending them to me as probable mitral stenoses. It so happened that most of them were what is now called, neurocardioasthenias. In other words, neuroses of the heart. Not a new condition, for in the Civil War it was known as "soldier's heart." The more recent verbiage was selected during the World War. In order to separate the neurocardioasthenic case from the true mitral stenosis, a history is essential. This particularly applies a search for rheumatic history on the one hand, or a neurotic personality on the other.

From a limited experience, and perhaps a little thought, I am inclined to leave this suggestion with you, that, in accord with the Mackenzie teaching, who, by the way, was rather a good obstetrician, most obstetricians stand in too much awe of their cases with heart complications.

OBSTETRICAL SOCIETY OF PHILADELPHIA

STATED MEETING, FEBRUARY 2, 1922

The President, Dr. Stephen E. Tracy, in the Chair

Dr. Ross McPherson, of New York, read, by invitation, a paper on **The Conservative Treatment of Eclampsia.** (For original article see page 50.)

DISCUSSION

DR. BARTON COOKE HIRST.—The speaker correctly states that no one single method of treating this condition will apply to every case and had I adopted a single method in all my cases in the last few years, I would have felt that I had

not given the patients a fair chance for recovery. I have used the morphia treatment for years, I believe in a rational manner. If a woman comes into the Hospital deeply comatose and remains so, with only one or two convulsions during her attack, it seems to me morphia would be inappropriate. If, on the contrary, convulsions are frequent and violent, morphia ought to be given in full doses to the extent Dr. McPherson advises, for unquestionably, the convulsions alone will kill the patient, but they are not the only cause of death. I gave up the exclusive operative treatment long ago, before 1909 I am sure, having had the same unfortunate experience with it that other observers have had. I think my clinic here was one of the first to abandon the theory that the main thing was to empty the uterus. Now one finds an agreement on this point everywhere. From an average death rate of close to 30 per cent with operative treatment as the main reliance, there is now in general an average mortality of 15 per cent or lower. But the operative treatment has a place in the therapeutics of eclampsia, and a more important place, I believe, than Dr. McPherson ascribes to it. Our utilization of the operative treatment is thus standardized: We apply what we believe to be all the rational methods and if they fail, then we turn to an operation as a last resource and we find that, preceded by elimination, sedative treatment, and all the measures to reduce blood pressure, it gives us surprisingly good results. If, indeed, we were to judge by these figures alone, we might claim that the operative treatment was the best for all eclampsia cases. In the last five years in the University Hospital we have not had a single death from it although we have only resorted to it in the worst cases. We have had in the five years preceding this, 89 cases of eclampsia: In seventeen instances cesarean section was performed without a death from the operation. There were two deaths following the operation, one three weeks later from staphylococcemia after the eclampsia had long ceased and the other from gangrene of the lung, also some weeks after recovery from the eclampsia and the operation. These deaths cannot be fairly ascribed to the operative treatment, but our mortality would have been considerable had we not prepared the patient to stand the operation by the treatment that preceded it. There is one treatment not referred to at all tonight. I understand the purpose of Dr. McPherson's paper perfectly, namely, to show the results that can be obtained by the sedative treatment alone; and they are most creditable, but I would like to emphasize the importance of the eliminative treatment, especially by the skin, a treatment apparently at present out of fashion. I heard Dr. McPherson refer to the eliminative treatment as excellent in the prophylaxis of eclampsia; if it is good in the pre-eclamptic, it ought to be good for the eclampsia itself. I think that a clinical observer with ordinary powers of observation, who sees this treatment properly conducted with a sweat cabinet, must admit it is efficacious; and it seems to have a logical basis. It is true that nobody knows what the toxins of eclampsia are or how they are eliminated, whether mainly by the kidneys, the skin or the bowels, but it is equally true that no one can say that the toxins are not eliminated by the skin, as well as by the bowels, the kidneys being temporarily out of commission. Every case of eclampsia, moreover, is secondarily a case of parenchymatous nephritis, with uremia superadded to the original toxemia. No one denies the utility of diaphoresis in the latter condition. The elimination by the skin also diminishes the hydraemia of pregnancy and, I think, lessens the danger of those multiple apoplexies, especially of the brain, so often found in a fatal eclampsia. Sweating also reduces blood pressure and thus is of value. The objection that it concentrates the toxins is met by routine proctoclysis alternating with the sweats. Like every one charged with the responsibility of a large maternity

hospital, I feel that the management of eclampsia is one of the most serious problems with which we are confronted.

DR. EDWARD P. DAVIS.—While we do not know the real cause of eclampsia, recent additions to our knowledge of its pathology cannot fail to be interesting and valuable. Since the close of the recent war the obstetricians and obstetric pathologists of England and Germany have been working anew at this problem. You will remember that some years ago it was stated that the toxemia of early gestation was caused by the absorption of fetal elements and there is much in favor of this view. Continuing this line of thought, a study of immunity and careful observations in microscopic anatomy and biochemistry have been made concerning the production of toxins by the placenta, the placenta containing as we remember, large quantities of fetal elements. It has been distinctly shown that from these substances are obtained certain toxins closely allied in chemical composition to the poison of certain serpents. One of them has an extraordinarily selective effect upon the cells of the liver and the parenchymatous cells of the metabolic organs, causing their destruction. One of these toxins attacks especially the substance of the kidney and these toxins exercise a most extraordinary effect upon the blood making and secretory glands of the body; the ultimate result of this action being the formation of minute emboli and thrombi carried by the toxin-laden blood extensively throughout the organism. This study links up the early involvement of the ovum with the toxemia of late gestation and gives the mechanical explanation of convulsions and also the explanation of the fact that eclampsia commonly ceases after the birth of the child. It is a matter of observation that within 36 hours after the uterus is emptied these trophoblastic elements cease to manufacture toxin and that is the reason why eclampsia ceases about that time.

Personally, I have always considered eclampsia as a case of toxemia and it has seemed to me that the primary factor in treatment was elimination and after trying all sorts of methods and having been one of the army of operating enthusiasts who gave it up, and I think obstetricians ceased to operate earlier than others, I have devoted my attention to ridding the blood of toxins and under no circumstances doing harm by a forcible method of emptying the uterus. So far as eliminative treatment is concerned, I am much in favor of venesection and the substituting of saline or glucose fluid and I do not take blood pressure as an indication for or against bleeding; the degree of toxemia, if I can make out, is the indication and the worst toxemic cases I see have a low blood pressure and not sufficient resistance to produce convulsions; and I would gladly take from them some blood and substitute a fluid which I hope will be of greater service. Elimination produced by gentle sweating, lavage of stomach and leaving in the stomach a laxative and copious lavage of the bowels and letting patient alone so far as the labor is concerned are all important factors in treatment. When she shows effort and desire to empty her uterus then help, if help be needed. At the same time I am in full agreement with Dr. Hirst that there come times when operation is indicated. For example, a stalwart young woman and fetus with good sound heart beat, is taken with a sudden outbreak of severe eclamptic convulsions. It is not quite time for labor and the cervix is tightly closed. Is it not then better to give her vigorous eliminative treatment and unless she immediately ceases to have convulsions, to operate, preferably an abdominal section, and empty her uterus? Should we not, too, carefully guard our patient's vision, and I am glad to observe Dr. McPherson's precaution in the matter of ophthalmoscopic examination. May I suggest that in my experience the removal of cerebrospinal fluid in patients who show beginning

changes in the retina is distinctly a conservative procedure and in our experience has greatly lessened the damage done to the eyes in these cases. Certain it is that prophylaxis must remain our great hope. Equally certain it is that wise conservatism has greatly lessened our mortality and morbidity.

DR. RICHARD C. NORRIS.—The diversity of opinions as to the etiology of eclampsia almost makes one believe that the convulsive seizures in pregnant women are perhaps analogous to convulsions in young children; that is, they are simply a symptom and that there is a multiform etiology. If the origin and qualities of the toxins, as pointed out by the reader of the paper, differ, we perhaps ought to concentrate our thoughts on that, and our treatment in some respects ought to be varied as the etiology seems to be. Surely with our lack of knowledge of what is going on in a pathologic way in our patient as the result of toxins, the origin of which we do not know and the destructive damages of which we cannot always predict, a uniform treatment as Dr. McPherson recommends cannot be equally efficacious in all cases. I am one of those who has never departed from the less aggressive form of obstetrical treatment. I believe that the effort to relieve the woman of the fetus, simply because we consider pregnancy the cause of her condition, is only applicable to the point when you see her approaching the danger zone. If pregnancy and eclampsia must be associated as cause and effect, the rational view to take of that phase of the subject is that the pregnancy should be ended before the patient is seriously damaged by the presence of the fetus and that dictum finds its true application in the induction of premature labor, when the removal of the fetus will do the most good. That argument has never appealed to me in the treatment of the patient actually eclamptic, whose liver, kidneys or brain may have widespread lesions that cannot possibly be restored by removing the fetus at so late a period of the disease. In the presence of an actually eclamptic patient, so far as our present knowledge goes, we only know and believe that she is a victim of a deadly poison of unknown origin. Our treatment should be concentrated on that idea and we may differ in our opinions as to the various procedures to secure elimination and combat the effects of that poison but if that is made the keynote of treatment we have gone as far as our knowledge warrants. In addition to that the stage of labor, the condition in which we find our individual patients, the clinical degree of the toxemia, the patient's history, the frequency of the convulsions and her mental condition between seizures, blood pressure, her cardiac action, her temperature, the kidney condition, and other clinical data, will influence the obstetrical and medical treatment.

I have treated some of my cases with cardiac stimulants rather than depressants, when even in the early stages the cardiovascular system has been almost overwhelmed by the toxemia. I believe I have saved life on many occasions by using caffein and even digitalis rather than depressants like veratrum.

As we study our cases clinically to appraise them as to the gravity of the toxemia so we must study them from the operative standpoint. Herein lies the strength of what several speakers have said. In a vigorous primipara, with undilated cervix, who has been treated by eliminative treatment, and in whom a prolonged labor is certain, cesarean section will probably be of advantage. If however, there are widespread destructive lesions, the removal of her fetus by cesarean section may come too late to save her. The same may be true of forceps delivery in multiparæ. The results of any operative delivery will depend more upon the degree of toxemia and its pathologic results than upon speed in delivery. As to eliminative treatment, you will find men varying in their opinions. Dr. Hirst has

pinned his faith to the exeretion through the skin. I consider purgation a more valuable means of elimination, not that I would neglect elimination from the skin, but in my clinical experience I have not seen a woman die from eclampsia where I have secured very free catharsis,—twelve or more evacuations within twenty-four hours and where the bowels did not respond I looked upon the patient as overwhelmed and the prognosis as almost hopeless. As to edema and the introduction of saline, I have been called in consultation to see women die because too much salt solution, without its elimination, has produced edema of brain and lungs. I think administration of salt solution, whether it follows bleeding or not, should always go hand in hand with the blood pressure apparatus. That is one's guide as to how frequently it should be repeated and with the degree of catharsis, the amount to be used is indicated. Now as to the use of bleeding; a pale, anemic, long continued toxic woman, with in some cases, a blood pressure which is below what you would expect in eclampsia, failing to respond to elimination and distinctly asthenic, is never benefited by bleeding, nor would the use of any of the cardiac or cardiovascular depressants like veratrum be indicated. As to the use of forceps, there is no question as to the advantage of terminating labor when that stage has been reached and I think we all agree to that. The real crux of the matter is, shall we consider speed in delivery as the first essential. If that ground be taken then cesarean section would be indicated for more cases. On the other hand if speed has no real value then all cases should be treated as outlined by Dr. McPherson. I find I am not in accord with either of those positions.

In a group of 23 cases of eclampsia in 5,000 consecutive patients at the Preston Retreat, which Dr. Bernard and I studied, and I reported in 1918, with two deaths, (8.9 per cent mortality), only one had rapid obstetric delivery, a case of vaginal cesarean section in a primipara. She died on the ninth day of pneumonia and the other woman died twenty-four hours after a simple low forceps operation. In that group there was no hurried obstetric treatment.

The outstanding fact from my experience in the "Retreat," is the experience gained from these cases in the pre-eclamptic stage. Treated by induced labor the convulsive outbreak is usually avoided.

We should urge the general practitioner to watch his cases and never let them go to the eclamptic stage. One of the striking results of the "Retreat" is, that we do not get the eclamptic cases that we did prior to the institution of the prenatal service. The real value of the prenatal clinic is the avoidance of neglected cases. To recapitulate, the eliminative plan of treatment is at the present time the best we know and the obstetric or surgical treatment of these cases must be determined by two factors, the clinical study of the individual case but from the standpoint of toxemic gravity and especially the appraisement of the soft tissues of the birth canal as an obstacle to the relatively early delivery. That helps me in my work more than any other two factors in deciding for or against a rapid delivery. Not knowing the actual degree of pathologic degeneration in the organs, a relatively quick delivery seems to me the wise course. It is not, however, a question of a few minutes rather than a few hours.

DR. WM. R. NICHOLSON.—I am still treating eclampsia in the same way that I have done all my professional life. I have never been attracted by the immediate operative delivery, but have always felt that elimination was the prime essential. The only exception to this plan in my practice, has been years ago, when the Bossi dilator first came into vogue. I am sorry to say that I did attempt to use it in a very few cases, but was immediately convinced that any form of

accouchement forcé was very ill-advised. At the present time, I am using more morphia than in the past, but I do not believe that the eliminative treatment should ever be made subordinate to the morphia except under conditions in which the eliminative treatment cannot be used for lack of proper nursing care. I particularly want to emphasize my thorough agreement as to the advisability of venesection and I am also greatly interested at this time in the use of sugar solutions intravenously. I think the work recently reported by Dr. Titus, while of course still not conclusive, is extremely interesting and well worth following out. I believe that eye-ground examinations as an index of interference in cases of toxemia, cannot be depended upon, as eclampsia attacks may occur before albuminuric retinitis is present and therefore, it is unsafe to wait for the development of this condition before inducing labor. In brief, I believe that at the present time, the proper treatment is the eliminative treatment for from six to twelve hours and then if the patient is not improving, it is necessary to take the chance of the operative delivery by whatever method is the least harmful to the woman, whether it be cesarean section, forceps or version.

DR. GEORGE M. BOYD.—It is as interesting to hear a conservative paper on eclampsia as it is to hear a conservative paper on any subject today in midwifery in this day of version as a panacea by one man and forceps another and cesarean section for eclampsia by the third. We have followed at the Lying-In-Charity of Philadelphia for thirty years the conservative treatment. We have been using morphia, elimination, blood letting and finally in the exceptional case resorting to surgery. The German method of using morphia in larger doses was instituted by Veit.

DR. WILLIAM E. PARKE.—I have recently reported thirty-six cesarean sections for eclampsia; so I should like to enter a plea in this meeting for this method of treatment in a certain group of patients. By this group I mean primiparæ who are not in labor, and who fall into convulsions before labor sets in. Those of you who have done these operations must, I think, have been impressed as I have, with the instant cessation of all convulsive phenomena as soon as the operation is done. Patients who may have been having convulsions at fifteen- or twenty-minute intervals stop abruptly. Only five of the thirty-six patients had a convulsion following the operation. Now I, of course, do not advocate cesarean section for every case of eclampsia, but for the primiparæ who have convulsions and are not in labor I think it is a worth while thing to do. Moreover, any conservative treatment you can employ without doing the operation, you can also employ with it; thus you have the added advantage of getting promptly rid of the cause, plus the conservative treatment.

Of the thirty-six cases I operated on, three died. One of these clearly should not be classed as eclampsia; it was a cardiorenal case with orthopnea and edema of the lungs in which the terminal event was a convulsion. Omitting this there were thirty-five cases with two deaths,—a percentage of 5.7.

DR. N. L. KNIPE.—I feel like answering Dr. Parke by saying that perhaps his good results from cesarean section were due to the letting of blood and in that way getting rid of a great deal of toxemia. I read a paper in 1916 on "The Treatment of Eclampsia", which gave very much the same results which Dr. McPherson has obtained. At the University, in Dr. Hirst's service, we carried out very much that same plan. Our chief eliminants are castor oil, if the patient is conscious, and croton oil if not; the sweating is simply a part in the whole of the eliminative treatment. We have always given morphia in severe convulsions. In 1916

I reported a mortality of six per cent in a series of 85 cases, including all except those dying within 24 hours, but including one dying a month after delivery.

DR. GEORGE CARSON HANNA.—In the discussion no one has spoken of the rights of the baby. Are there not cases, especially in young primiparæ, where the birth canal is not easily distensible and the cervix remains hard and elongated, where more radical measures would not increase the maternal mortality and yet get a living baby? If these are left to Nature, they often drag along for several days with its attendant danger to the fetus. While my experience is not as large as some here, yet I feel after a series of a hundred cases that one can be too conservative.

We are now in the treatment of this disease about where I started twenty-five years ago. Then it was watchful waiting, then came the era of operation and radical treatment. Once more the pendulum is turned backward, and now non-interference again is becoming the watchword. The sooner we realize that each case should be individualized and the personal equation of the individual taken into account, the better will be our results. For what would be good procedure for one might be highly improper for another.

DR. ALICE WELD TALLANT.—Everybody uses morphia, everybody operates when necessary and everyone does venesection. We individualize our cases and that after all is the only thing we can do. We cannot lay down a method of treatment that will suit them all. There was one class of cases not mentioned, the cases of postpartum eclampsia. Perhaps I have had very bad luck, but some of our worst cases at the Woman's College Hospital have been postpartum cases in which the question of delivery had no place. For a patient already delivered, we could not do cesarean section, and treatment by elimination, morphine and so on proved futile at times. It is usually stated, I think, that these cases are more benign than those occurring antepartum, but in my experience postpartum eclampsia has been so severe that if any suggestion could be added to help in the treatment of these cases I should be glad to hear it.

DR. DANIEL LONGAKER.—I would like to add one thought to this discussion. I think we should use a conservative rational treatment rather than the radical treatment of convulsions. As the last speaker has said, we all treat our patients very much in the same way. If they are efficiently treated conservatively I believe that a very large number of potential eclampties will be cut out. I want to make a plea for compound jalap powder for elimination and I believe elimination by the bowel is safer. It may be a little disagreeable, but it is perfectly safe and efficient. The point of the hydremic character of the blood has been brought out and this hydremia is lessened by the jalap powder. Another point is the constant presence of acetone and diacetic acid, in other words, acidosis, in these cases. This gives a very valid ground for the application of alkalies which are certainly efficient in reducing tension. My colleague, Dr. Parke, is exceedingly modest and I think he has not said all that he could have said in favor of radical, or operative treatment. Over at the Kensington Hospital it is certainly a very popular plan and I think if we did not resort to it that frequently we would have to offer some explanation. The preceding speaker has said something about the rights of the baby. We do know that persistent convulsions treated conservatively result in a large fetal mortality.

DR. McPHERSON, (closing).—There are one or two things which impressed me very markedly and the first is that the Philadelphians are so universally ahead

of us in New York, because I assure you in New York the operative idea prevailed and has until comparatively recently. I thought I was presenting something reasonably new, although we have recognized Dr. Kniple's work and it has been stimulating to us for several years. Some of the younger members of this Society have had service under me in the Lying-In Hospital and Dr. Neely has had four cases successfully treated conservatively but lately he has fallen into the by ways and done cesarean sections. Regarding the statistics on cesarean section for eclampsia from all over the country, if they are taken and digested, notably a series of cases by Dr. Reuben Peterson, of Ann Arbor, in which he reported 500 cases of eclampsia treated by section with a high mortality, the figures from the clinic in Glasgow where they do a very large number of cesarean sections and the report from the Sloan Maternity Hospital showing again a high mortality, gives us an indication regarding the position of this operation. I must congratulate you gentlemen who can present a mortality of zero on Dr. Hirst's service up to about 9 per cent on Dr. Parke's. I say this wholly in the spirit of appreciation and I do not mean to be sarcastic but once when I reported 40 cases of eclampsia with a mortality of about 7 per cent, a colleague said, "When the Doctor has had more cases he will have a higher mortality" and I am wondering if in Dr. Parke's series he has run into his lucky streak, combined with his skill in operating which has made his results successful. I believe we do run into these streaks in eclampsia. I had a large series of cases which ran along without any deaths at all and I was convinced I had solved the problem. Then I had three deaths in succession. I agree thoroughly with the statement that there are some cases in which cesarean section is indicated; I myself did one several weeks ago on a young primipara who had a long, undilated cervix, who was at term, with the pelvis not any too large and it was highly essential to secure a living baby. At the same time I cannot feel that it is the indicated operation in the majority of cases. It seems to me from the general discussion that the subject divides itself into two things; that you all approve of either letting eclampties alone, or operating on them, and I will agree with that. Then the next thing is that question of elimination. It interested me very much to hear what Dr. Hirst said, but I am somewhat disappointed because I have not had the same result. I gave up sweating my patients ten years ago because I lost so many of them in the pack; after all the skin is the poorest means of elimination we have. It does not eliminate anything but water in any marked amount and by this means you concentrate the toxins in the system instead of diluting them, which is not what we are after. As I have talked it over with men all over the country, the results obtained have been pretty generally in favor of discontinuing sweating. I have seen patients getting on pretty well, put in hot packs and then die. I am afraid of it, and it is not an isolated opinion upon my part. On the other hand, I realize that we oftentimes see results in some places which are very different from what they are in others. In so far as the blood chemistry is concerned, I did not go into that purposely, for we are still undecided as to what blood chemistry means and so far we have not been able to arrive at any conclusion that gives us any help. Acidosis apparently plays a very small part in the condition. There is an article in *The American Journal of Medical Sciences*, where this subject is very carefully worked out by Losee and VanSlyke and they showed that the part the acidosis played was very slight. In regard to transfusion with saline after bleeding, I have discontinued its use and I am inclined to believe that you can get as much good with large high colonic irrigations. By catharsis and irrigation I tried to convey the idea that I approved of these procedures in the article. Concerning two more things that were

brought up; first, the question of saving the baby. Of course there is not anybody more willing to admit that the baby ought to be saved than I am. After all that is the aim of the whole process not only to get a living baby but also to get a living mother if possible. It is a matter of fact in no case in which a fetal heart was heard at the time of admission in this series was the baby stillborn, except one and that had a cord complication which would have resulted fatally under any circumstances. Of the other babies that died there was no evidence of life at the time of their entrance into the hospital; and as to the question of long labor, as a rule the labor was not long, for after morphinization the labors were extremely rapid. The children were not subjected to anything like the trauma, or pressure that they would have been if the mother had been operated on. In regard to the treatment of postpartum cases, I regret that I cannot at this moment tell you just how many postpartum cases I have had, I agree that postpartum eclampsias are much more serious than antepartum, and our figures show that. It naturally follows that if pregnancy is the cause of the trouble and delivery does not relieve the patient that the condition must be severe. I have treated them in the same way by trying to control the convulsions and trying to assist elimination and have been reasonably successful.

DR. E. A. SCHUMANN AND DR. HARRY S. FIST (by invitation) read a paper entitled **Organic Disease of the Nervous System Complicating Pregnancy with a Report of Two Cases.** (For original article see page 67.)

DISCUSSION

DR. EDWARD P. DAVIS.—This paper is germane to the subject of the evening, eclampsia and toxemia of pregnancy. We recognize, as has been said by the readers of the paper, that toxemia is the cause of neuritis and the cause of many of the peripheral nervous diseases in the cutaneous system, and also the cause of certain spastic and toxic conditions by virtue of multiple emboli of the nervous system. There is one disease of the nervous system, which is comparatively rare, fortunately, namely, acute meningitis accompanied by acute insanity. It is almost universally fatal and often in the acuteness of her insanity the mother does violence to the child. The mother invariably dies and the infant is rescued with the greatest difficulty. In nervous lesions complicating gestation one must always think of the presence of syphilis and the Wassermann reaction must be taken and all means taken to ascertain the truth.

DR. BARTON C. HIRST.—As a commentary on the induction of labor in these cases I recall one in which the termination of pregnancy had no effect upon the mother whatever, the myelitis steadily ascended until the woman died.

DR. ALBERT B. DAVIS, Camden, N. J.—A case of acute transverse myelitis which I saw, occurred about fourteen hours after delivery in a primipara, with a perfectly normal labor. I did not see very much of her before she came for delivery, but the only thing at all abnormal that I know about her is that she seemed to have a little uterine inertia, she was a rather weak woman and I gave her one-third of 1 c.c. of pituitrin and she had her baby in about twenty minutes after that without having any laceration or any trouble. She had a good night, and I saw her the next morning at about 11 o'clock. Just before I arrived, she complained of some pain through the shoulders and while I waited she was completely paralyzed from the sixth cervical vertebra down. Dr. Spiller saw her in consulta-

tion. The Wassermann tests of her blood and spinal cord and of her husband's blood were negative, and Dr. Spiller said he supposed probably the pituitrin had something to do with it, possibly in some way causing a thrombosis in the vertebral artery, which I think was about as far as we could ever find out. Of course it came on too soon after labor for infection from the uterus. She lived about six weeks and never recovered any sensation. She breathed entirely with the phrenics and diaphragm. There was no postmortem permitted.

GEORGE ERETY SHOEMAKER, M.D.

JANUARY 5, 1922.

In the death of Dr. George Erety Shoemaker, the Philadelphia Obstetrical Society has suffered a great loss. Dr. Shoemaker had been a member of this Society for thirty-three years, having been elected in 1887. His active interest in our scientific business is well attested by his frequent contributions to our transactions, as well as by his regular attendance at our meetings during many years. The last paper ever prepared by him was to have been presented to us last month, but his death occurred very suddenly upon the day of the meeting.

In view of Dr. Shoemaker's long service in the halls of the Obstetrical Society, it is fitting that commemorative action be taken and therefore, your Committee offers the following:

> "WHEREAS, in the death of George Erety Shoemaker, the Obstetrical Society of Philadelphia has lost an active and enthusiastic member of high ideals, broad charity, and matured attainments; therefore, be it resolved, that a copy of this minute be sent to the family of the deceased, as a manifestation of our sympathy in their bereavement and that it be spread upon the minutes of the Society as a mark of our respect and sorrow."

WM. R. NICHOLSON,
JOHN H. GIRVIN,
COLIN FOULKROD.

NEW YORK ACADEMY OF MEDICINE

SECTION ON OBSTETRICS AND GYNECOLOGY

STATED MEETING, MARCH 28, 1922

DR. WILLIAM P. HEALY IN THE CHAIR

DR. A. J. RONGY reported a case of **Fibroma of the Ovary Complicating Pregnancy.**

Mrs. H., admitted to the Lebanon Hospital, Feb. 3, 1922, twenty-two years of age, white, married, primipara, born in Russia. I was called to see her in consultation during labor, and found a thick, conical cervix dilated about one and one-half fingers. In addition I felt a hard, irregular tumor obstructing the lower three-quarters of the pelvic outlet. The tumor had the general consistency of an exostosis. A cesarean section was done after which I removed the tumor which was

very hard, originating in the ovary and having a pedicle consisting of a sheath containing parovarian structures. The lobular tumor measured 15x9x5 cm. There were two distinct parts seen on section, an outer zone measuring 3 cm., hard and solid, and an inner cystic area. There were no gross evidences of an acute inflammation. On cross section, the pedicle showed three zones: an inner whitish zone similar to the tissue of the tumor, a middle mucoid zone, and an external thin tunica which limits the pedicle. This latter is apparently composed of connective tissue. Microscopically, some areas show nothing but firm connective tissue. Other sections show pictures similar to that of pedicle. One area shows a definite corpus luteum cyst. No evidences of malignancy. Microscopically, the pedicle presented an external zone of connective tissue cells making a rather definite tunica. Within this there was a loose structure of fibroblastic cells. Throughout this section there are numerous small cystic areas, a few lined with a deep layer of irregularly arranged epithelial cells. The nuclei of these cells are poor in chromatin. Some cysts are lined with a zone of connective tissue very poor in cellular elements. Tissue is rather sparse in blood vessels. Diagnosis: Fibroma of ovary.

I present this case to the Section for the purpose of evoking a discussion as to the proper procedure to follow in a case of tumor complicating pregnancy at term. Shall we remove the obstructing tumor and perform cesarean section or shall we simply remove the tumor and allow the patient to deliver *per via naturalis?*

DR. RONGY also reported a case of **Hernia of the Pregnant Uterus with a Fourth Cesarean Section.**

Mrs. L., admitted to the Lebanon Hospital, Oct. 20, 1921, thirty-five years of age, white, married, primigravida. Her gestation period at the time of admission to the hospital was seven and a half months. She presented a very marked median ventral hernia, through which the gravid uterus protruded covered only by skin and subcutaneous fat. The skin had given way in the upper part of the hernia with a resultant ulcerated and granulating area. The patient was put to bed immediately on admission but despite all treatment the ulcerated area increased in extent. I had previously performed three cesarean sections on this patient. She refused to be sterilized always hoping that the next child would be a boy. I waited until term and performed the fourth cesarean section, sterilized her and repaired the hernia. Patient made an uneventful recovery.

The reason I present this case is to get the sense of the members of the Section as to the best procedure. Should this woman have been aborted in the early months of pregnancy or should her case have been managed as it was?

DISCUSSION

DR. EDWARD W. LEE.—I think no adverse criticism can be made on the procedure in the first case.

So far as his second case is concerned, I believe that woman was entitled to have her uterus emptied. She must have had wonderful care to have gone through so much, with the hernia and ulcerated abdominal wall. I condemn his procedure of allowing her to go through so much suffering, and I think Dr. Rongy should have emptied her uterus.

DR. FREDERICK C. HOLDEN.—I believe that in such cases the course to be followed depends on: (1) the condition of the patient; (2) the condition of the child, and (3) the amount of dilatation of the cervix. I cannot see any reason

if the woman is in good condition and the child in good condition, why it is necessary to do a cesarean section. I think one must be governed entirely by individual circumstances in each case. As for the second case, I think that when a woman has had two cesarean sections she is entitled to sterilization.

DR. HEALY.—It seems to me the results in these two cases justify the procedures carried out by Dr. Rongy.

In regard to the second case, I should have disliked to induce an abortion just because the patient had a hernia of the uterus through the abdominal wall. I think that Dr. Rongy's method of procedure was the proper one. He took good care of the patient and showed that a woman could be carried through pregnancy and could then be cured of her hernia, and therefore an abortion would not have been justified. I think, as Dr. Holden says, that the woman should have been sterilized by ligating the tubes at the time of the cesarean section; but to interrupt pregnancy would not have been justified.

DR. RONGY.—In reply to Dr. Holden, I would say that in this case the cervix was just beginning to dilate; one could not tell how long it would take the patient to complete her first stage. Therefore I felt that it would not be advisable to allow the woman to go through her entire labor, immediately following a serious abdominal operation, and I incised the uterus and removed the child. The only objection to an abdominal delivery, in a case of this kind, is the scar in the uterus. However, weighing both questions in my mind at that time, I decided in favor of the latter.

As to sterilization: many women are prejudiced against sterilization; there is a notion among some of them that if they are sterilized, they become sexually unfit, and it is more on this account that they object than because they will not be able to bear any more children. I agree with Dr. Lee that fat women particularly, who have had two or three cesarean sections, should not be subjected to repeated operations. These women should be told of the danger and some of the complications that may accompany multiple sections.

DR. HENRY FURNISS reported a case of **Supernumerary Ovary.**

This patient, thirty years of age, was seen on April 25, 1921. Menstruation began at the age of thirteen, of the four-weekly type, five days' duration, always attended by crampy pains in the lower abdomen, and was profuse until she was twenty-five; since then the flow has been moderate.

When eighteen years of age she began having pain in the lower abdomen, in the center and the right side. At first this came every four weeks, lasting a day or two, but not at the menstrual period. During the past year the pain had become worse, and for three months had been severe and almost continuous. In December, 1919, she had influenza pneumonia, and since then has been rather frail.

Examination showed a mass the size of an orange in the pelvis, posterior to the uterus, cystic and very tender.

On April 30, 1921, a median abdominal incision was made. A mass 10x7x3 cm. was found, attached by a pedicle, one-eighth of an inch thick by three-fourths of an inch broad, to the upper posterior surface of the uterus, just internal to the tube, and adherent to the culdesac. This was removed by division and suturing of the pedicle. Two normal ovaries, normally situated were found. Recovery was uneventful.

Pathological Report: A rather thick-walled cyst contained chocolate colored, broken down, opaque fluid material. The wall is from 5 to 12 mm. in thickness and appears to consist of edematous fibrous tissue. On further sectioning, serous cysts, from 5 to 8 mm. in diameter were discovered in the substance of the wall. Sections of the cyst wall show in part a lining of columnar cells covered with blood clot at certain points and in part, denuded areas with the exposed stroma filled with blood pigment. The wall is dense, fibrous tissue infiltrated with round and polynuclear cells beneath the epithelium. It is somewhat edematous; contains large blood vessels; many small capillaries with round cell infiltration about the vessels and polynuclear cells in the lumina of many. Blood extravasation and blood pigment is common in the wall. There is a small cyst in the wall lined by several layers of flattened cells that seems to be a dilated graafian follicle.

Diagnosis: Ovarian cyst showing chronic inflammation and degeneration with small cysts in the wall.

This is a true case of third ovary with cystic degeneration.

DR. HARBECK HALSTED reported a case of **Morphine Poisoning of the Fetus Before Delivery.**

This case is of interest because it is one of profound poisoning in the infant from morphine administered to the mother before delivery, followed by recovery after treating the infant along the lines advocated for morphine poisoning in the adult.

The mother was colored, eighteen years of age, a primipara. The probable date of confinement was Feb. 2, 1922. The patient was first seen in the prenatal clinic Dec. 12, 1921. At that time she gave a history of vomiting twice early in pregnancy, none since. Her appetite was poor; she was constipated; and complained of occasional headache and slight burning on urination.

She was seen in the antepartum clinic at regular intervals. Her blood pressure was never above 118/65 and her urine was negative. The diagonal conjugate was 10 cm.

On January 24, 1922, she was admitted to the Sloane Hospital for observation. A consultation was held and a cesarean section considered as probably the best method of delivery. She developed a cold with a cough so the operation was postponed from time to time waiting for it to clear up. By some oversight the blood pressure was not taken and the urine was not examined at regular intervals. During the night of Feb. 13, 1922, she was wakeful and the morning of the fourteenth complained of a severe headache. She was given aspirin 5 gr. at 2:30 A. M. and again at 11:45 A. M. Feb. 14 she had a profuse bleeding from her nose, vomited some old blood and complained of severe headache, dizziness, and spots before her eyes.

On examination she was found to be very toxic, nervous, to have edema of the legs, reflexes markedly exaggerated; her blood pressure had risen to 142/80 and her urine showed albumin .607 by gravity. It was decided to put her on routine treatment for marked toxemia, which is to keep the patient deeply under the influence of morphine in addition to other treatment.

It was also decided that as soon as the patient was well morphinized a cesarean section should be done as the only method of delivery holding out any hope for the baby.

She was put on a low protein, salt-free diet, forced fluids and absolute rest in bed. At 12:10 P. M., Feb. 14, she was given morphine sulphate ¼ gr. by hypoder-

mic; at 8:30 P. M. chloral hydrate gr. XX and sodium bromide gr. 15 was given per rectum. At 11 P. M. she had another epistaxis which lasted ten minutes and was followed by the vomiting of old blood. At 2:15 A. M., Feb. 15, morphine gr. ½ by hypodermic was given. At 3:50 A. M., she vomited a small amount of coffee ground material. At 4 A. M., morphine gr. ¼ was given by hypodermic. At 5:20 A. M. the patient coughed up clotted blood and vomited a dark red fluid. She rested fairly quietly after 2:30 A. M. At 9:05 A. M. she vomited dark brown fluid and was given gr. ½ by hypodermic. At 10:05 A. M. she was again given morphine ¼ gr. by hypodermic. She was also given a colon irrigation. At 11:05 respirations were 11 per minute; at 12:45 P. M., respirations had fallen to 6 per minute. At 2:55 P. M. she was delivered by cesarean section of a baby girl weighing 3629 grams.

In all our previous toxic cases treated with large doses of morphine the effects had apparently worn off during labor. The baby's heart was normal but it was hard to get her to breathe. After about thirty minutes of tubbing in hot and cold water with artificial respiration the baby cried, but on arrival in the nursery a few minutes later she was very blue, the respirations were very shallow and had nearly ceased; when spanked and aroused the color and breathing immediately improved. Oxygen was administered but did not seem to do much good. As soon as the baby was left alone she became cyanosed, the respirations slow and shallow and once ceased entirely; this time she was given a mustard bath which again revived her. During this bath, about an hour after birth it was noticed that the pupils were pin point and that the respirations reacted to stimulation as in morphine poisoning. It was determined to treat the baby as an adult who was so poisoned. Atropine sulphate, gr. 1/600 per hypodermic was repeated twice at three-hour intervals. About ten minutes after the first dose of atropine, adrenalin 1-1000, min. 3, and caffeine citrate, gr. ⅓, hypodermically; gastric lavage with potassium permanganate solution about 1-100,000. Coffee ounces one per rectum.

It was necessary to arouse the baby at frequent intervals for nearly eight hours and a second mustard bath was necessary about three hours after birth. For three days after birth the baby vomited after each feeding and even vomited after gavage—probably as an after result of the morphine poisoning. After the first three days the baby's progress was normal.

DISCUSSION

DR. HARRY ARANOW.—While I have never seen a case of morphine poisoning in a baby to the extent of that reported by Dr. Halsted, I have seen many infants slightly affected by narcotics administered to the mother. When we worked with "twilight sleep" some years ago I saw children born with pin point pupils and partly narcotized. In the majority of the cases the influence of the morphine gradually wears off without the adoption of any very drastic treatment. The mother in this case I believe was not excreting her morphine in a normal way as is indicated by the fact that her respirations kept going down. She retained the morphine for more than four hours.

DR. ROYAL C. VAN ETTEN.—We have in ordinary cesarean sections done away with the use of morphine and atropine before operation. For a long time we have noticed that the babies taken from mothers who had these previous injections, which were within one-half hour before anesthesia was started, did not do so well.

DR. HOLDEN.—Several years ago I did a cesarean section for dystocia. She was given 1 grain of morphine. The baby was somewhat narcotized and became

very cyanotic, so that it could not be left alone. We placed a small rubber catheter in the baby's nose and held it in place with adhesive plaster, and administered oxygen. The irritation of the catheter and the oxygen was all the baby needed. After twenty-four hours the catheter was removed.

DR. HALSTED.—We tried to give oxygen in the ordinary manner, but the baby did not breathe or the breathing became very shallow and at one time stopped altogether so the oxygen was not absorbed. The interesting point is the fact that we treated this baby exactly as the text-books recommend treatment for adults and with success.

DR. SINCLAIR TOUSEY reported a case of **Fibroma of the Uterus Cured by Deep X-Ray Therapy. No Return of Tumor in Three Years.**

The patient Mrs. D. referred by Dr. T. Spenser Halsey about four years ago, was extraordinarily large but refused to be weighed, and was 38 years old. She had had a fibroid for 4 or 5 years, abdomen somewhat enlarged. She had very severe hemorrhages, from which she was sometimes almost exsanguinated and on various occasions packing had been employed. She had a weak heart and was very nervous. She had been treated with radium, seven hours, at one of our best institutions, but this had not caused her to miss even one period. The details of the radium treatment follow:

"May 1st, 1918, radium was inserted into the uterine canal for 677 mc. hours, screening being 1 mm. platinum and 1 mm. rubber. May 30, 1918, she was given an external treatment over the tumor at a distance of 4 cm., area covering 30 square cm., screening ½ mm. platinum, for 3100 mc. hours."

There was a fibroid as large as a grapefruit of the body of the uterus and two in the cervix the size of walnuts were visible with a speculum, as well as palpable.

All the x-ray treatments were with my own generator which delivers a constant, unidirectional voltage at the terminals of the x-ray tube, the result being a stream of cathode particles starting under the influence of a uniform voltage, the object being to secure x-rays of approximately the same velocity, which many different tests have shown is approximately the case. So that with a comparatively light filter, thereby wasting only a comparatively small part of the radiation, I am enabled to produce a sufficiently deep effect, usually without even reddening the skin.

Wintz and Seitz have not had the opportunity to employ such a constant current but state their belief in it. Their own work has been done with a voltage of 160,000, consisting of a series of waves from a very low to a very high voltage. They agree that the very high voltage does not generate any x-rays of greater penetration than the more penetrating rays generated by moderate voltages of the ordinary impulsive type. But the higher the voltage the greater is the percentage of the more penetrating rays. Comparative tests have shown that with the ordinary type of generator even 160,000 volts do not give as great a percentage of the more penetrating rays as a constant current with 95000 volts.

The applications in this case illustrate the effects of different doses of radium and the x-ray in this condition in a large woman with a fibroid as large as a grapefruit. 677 millicurie hours intrauterine had no effect, neither did 3100 millicurie hours externally.

Of my own x-ray applications the first consisted in 10 minutes, 83 kilovolts, 3 ma., 10 inches, 3 mm. aluminum over each of six areas, *a*. upper, *b*. lower part of right lower quadrant of the abdomen; *c* and *d*, upper and lower part of left lower quadrant of abdomen; *e* and *f* right and left sacral regions. This was followed by amenorrhea lasting one year. The next series was as follows: the same areas as above except that the four quadrants of the hypogastrium received an additional dose a month later. Besides this the voltage was 87 thousand instead of 83 thousand. The time of exposure was the same except that for each of the two sacral areas it was only 5 minutes instead of 10, and for these the filter was 1/5 mm. instead of 3 mm. aluminum.

After this series given in May, 1919, there was immediate and complete amenorrhea and complete disappearance of all the fibroid tumors. The patient felt much less nervous than before. A couple of years later she was married again and

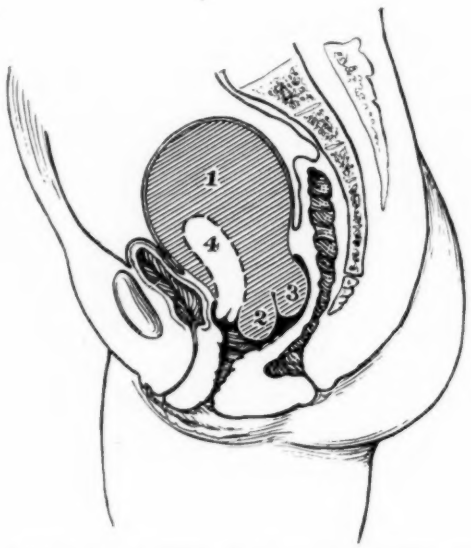

Fig. 1.—Diagram showing *1*, fibromyoma of uterus; *2*, *3*, tumors visible with vaginal speculum; *4*, uterus of normal size. Tumors and hemorrhage cured.

found she had normal sexual desires and gratification. Three months later she was terrified to find perfectly normal menstruation occurring. There was no recurrence of the fibroid tumors but the fear of a return of the dreadful hemorrhages made her very nervous. I persuaded her to wait for another menstruation but then she said she was leaving for the country and Dr. Halsey and I both felt that another course of treatment with a possibility of again producing amenorrhea would be preferable to the terror she was laboring under.

At this time the treatments were with a 10-inch spark, 2 ma. 3 mm. aluminum, 11 inch distance, four areas in front and two behind treated for 10 minutes each, avoiding a part of the right sacral region where an almost imperceptible net work of cutaneous vessels outlined one of the previous x-ray applications.

The treatment at this time was not given because of any return or prospect of return of the tumor or hemorrhages but simply because of the patient's absolute terror.

DISCUSSION

DR. EDWARD W. LEE.—I know that you will criticize me, but I cannot understand why that woman was not operated upon. Here were three fibroid tumors, one as big as a grapefruit and why was she treated with radium and x-rays when even in ordinary hands, hysterectomy is so very satisfactory? I have performed many hysterectomies and I have yet to be persuaded that radium and x-rays are the treatment when even in ordinary hands hysterectomy is such a safe and sure procedure. I will not go into the possibilities of the presence of malignancy in connection with fibroids, but here was a case treated indefinitely and she does not know whether she will have to return for further treatment.

DR. TOUSEY.—This report was not intended to show which cases should be selected for radium, which for x-ray treatment, and which for operation. It was simply to report a case selected for this form of treatment on its own merits. From the size of the tumor it was not adapted for radium treatment but proved to be favorable for x-ray therapy.

An operation either from viewpoint of the work and expense required by the surgeon and nurses or from the viewpoint of the patient's suffering and disability is a very different matter from forty-five minutes' x-ray treatment on three occasions in four years.

Dr. Van Etten in his paper referred to Dr. Kelly's statement that in young women with severe hemorrhage he preferred to curette twenty times rather than to use radium. The reason Dr. Kelly made that statement was not that the radium was not effective in controlling the hemorrhage but because it might sterilize the patient and interfere with sexual functions.

(*To be continued in the August issue.*)

ST. LOUIS GYNECOLOGICAL SOCIETY

MEETING OF FEBRUARY 23, 1922

DR. E. LEE DORSETT, CHAIRMAN

DR. HUGO EHRENFEST presented a paper entitled **Better Obstetrics and the Problem of the Birth Injuries of the Newborn Infant.** (For original article see page 61.)

DISCUSSION

DR. GEORGE GELLHORN.—This paper has made me realize how little attention, on the whole, I have paid to the subject of birth injuries and how many problems of practical obstetrics should be viewed from this new angle. The grosser, more obvious, traumatisms we have always taken into consideration. I add to Dr. Ehrenfest's list the cases of torticollis of which I have observed two within the last year. One was a true torticollis, due, probably, to a persistent faulty attitude of the fetus *in utero*. An asymmetry of the face further testified to the congenital origin of the anomaly. The other case was one of pseudo-torticollis caused by an hematoma into the sternocleidomastoideus muscle during the breech extraction of a very large child. Complete restoration was obtained in both cases.

DR. L. M. RIORDAN.—It will be interesting to mention in this connection the result of postmortem examinations made in Dr. De Lee's service at the

Chicago Lying-in Hospital. We discovered injuries of some sort in about 70 per cent of all the necropsies, of which between 40 and 50 per cent were brain lesions.

DR. FRED J. TAUSSIG.—I will ask Dr. Ehrenfest to answer a few questions: Would a systematic radiographic examination of the newborn seem desirable? Are the methods of ascertaining the clotting time simple and accurate enough to be extensively employed? With the fetal head deep in the pelvis, would forceps or pituitrin seem more preferable?

DR. PERCY H. SWAHLEN.—I think that many other causes, especially syphilis, are more likely to be responsible for idiocy and other mental defects than a birth injury. In one of my cases, a forceps extraction, the larynx had been injured by a cord tightly coiled around the neck. The condition was considered an asphyxia and the efforts at resuscitation naturally proved a failure.

DR. OTTO SCHWARZ.—The babies' ward of the obstetrical department of the Barnes Hospital is daily visited by a member of the pediatric department. The determination of the blood clotting time is a routine carried out on every newborn.

DR. R. E. WOBUS.—Unnecessary haste apparently is one of the more common causes of serious injury of the infant. A radiographic routine examination of the newborn, as suggested by Dr. Taussig, would imply a definite danger since it would mean too large a dose of rays, but certain regions, supposed to be injured, could be easily studied under x-rays. As regards cranial indentations and fractures it would seem the safe rule to operate whenever symptoms of an intracranial hemorrhage are evident.

DR. HENRY STORRS.—Is there any connection between cephalhematoma and intracranial hemorrhages?

DR. RANDALL S. TILLES.—I wish Dr. Ehrenfest would explain why he assumes a relation of twilight sleep to intracranial lesions.

DR. EHRENFEST (closing).—It would be impossible to answer all the questions I have been asked. This would take more time than I devoted to my paper written for the purpose of awakening in you, as obstetricians, appropriate interest in two features of the problem of the parturitional injuries of the child, their prevention and early detection.

Haste is a significant factor in their causation, and especially if this haste leads to the quick and forced compression of the head. The expulsion may be dangerously hastened by a large dose of pituitrin, by the application of the forceps, or by too quick an extraction of the aftercoming head in the unjustified fear that otherwise the child would succumb to the compression of the umbilical cord between skull and pelvis. These are the most common conditions which supply the one factor of quick compression of the head. The other element, that of excessive compression, is furnished by a pelvic contraction, by a faulty technic in the extraction of the advancing head by means of the forceps or of the aftercoming head in a breech labor if the operator fails to bring, in accord with the mechanism of labor, the various diameters of the head in proper relation to certain pelvic diameters, because even in a normal pelvis, too large a head diameter is forced through relatively too small a pelvic diameter. Excessive compression of the small but soft head of the premature infant might be accomplished by its forced passage merely through an incompletely dilated cervix or rigid vulvar ring. The intracranial injury may be slight and again it will be haste in resuscitation which will be the cause of further traumatization, aggra-

vating the primarily slight intracranial lesion and adding other injuries. In the practice of obstetrics surely experience should result in less haste and more deliberation.

In our present state of information concerning the causation of intracranial injuries the choice between pituitrin and forceps should be determined by the decision in the particular situation whether the one or the other is less likely to cause either a quick or an excessive compression of the head, and under such conditions a liberal episiotomy or a preliminary ironing out of the vagina, as taught by Potter, might eliminate a definite danger to the child.

At least for theoretical reasons twilight sleep is objectionable as far as injuries of the child are concerned, because it delays the second stage of labor, very often leads to the necessity of employing pituitrin, probably in 85 per cent of the cases requires termination of the labor by forceps, and in a considerable number of cases makes artificial respiration necessary, all factors of recognized significance in the causation of intracranial injuries. In individual reports of cases one meets often with the assertion that "twilight sleep" was more or less directly responsible for the fatal injury. Some neurologists predict that it will take many more years to realize all the damage done to the baby's brain by "twilight sleep." Personally I feel that in some cases, in which the obstetrician uses enormous doses of morphin and scopolamin to keep the given promise that the patient will feel no pain, aside from a possible mechanical injury, a chemical injury might also be inflicted on the infant.

Rodda's method of ascertaining the blood clotting time is not accurate but simple, and entirely adequate for practical purposes. Whenever this simple test only suggests a delay of clotting it will always be advisable to resort to the entirely harmless procedure of injecting whole blood of father or mother subcutaneously. This is not only in itself a very good curative procedure but of great prophylactic value if further symptoms should make surgical interference necessary.

Radiographic study of a certain region is requisite for the correct diagnosis of certain injuries, e. g., fracture of the clavicle, differentiation between brachial palsy and detachment of the upper epiphysis of the humerus, or recognition of an injury of a cervical vertebra which may only simulate a torticollis, etc.

Department of Reviews and Abstracts

Conducted by Hugo Ehrenfest, M.D., Associate Editor

Collective Review

The Kidney of Pregnancy

By Edgar F. Schmitz, M.D., St. Louis, Mo.

IN REVIEWING recent literature on the so-called kidney of pregnancy, with its associated conditions of edema and hypertension, one is struck by the variety of classifications of kidney disease encountered, and the rather confusing nomenclature employed. For our purpose it is not essential to minutely examine the foundations on which these various overlapping and often conflicting divisions of pathology and symptomatology are based. It will suffice here to bring out the more salient points of those facts which seem fairly well established.

In studying renal involvement during gestation one must necessarily differentiate a true nephritis from those nephropathies under discussion, for upon this differentiation depends our future attitude on what may be called the kidney of pregnancy. Any case in which renal changes appear prior to conception cannot strictly be classed with the type of disease under discussion, even if the symptoms are aggravated by the subsequent impregnation. Nor should a case in which permanent renal damage is found following delivery be placed in this category, for v. Leyden long ago pointed out that one of the characteristics of the true nephropathies of pregnancy is the rapid disappearance of all symptoms following the emptying of the uterus by natural or artificial means. Heyneman[10] and others have recently again emphasized a point in differential diagnosis, by calling attention to the fact that the kidney of pregnancy is manifestly based primarily on degenerative processes, while a true nephritis is primarily of inflammatory origin. It is true that borderline types will be encountered now and then, where it will be difficult to determine which condition dominates the clinical picture, but in the main the kidney of pregnancy does not show the changes found in nephritis.

The newer views on this subject are taking a much broader interpretation of the symptom-complex exhibited, and have followed the line of reasoning so ably stated by Atchley[1] in the concluding paragraph of his article, in which he says, "One must express the feeling that the investigation of this disease (nephritis) has been hindered by an interest too closely restricted to the kidneys. A broader study of the chemical balances of the body as a whole may demonstrate that the kidney is of secondary importance, both from an etiologic and pathologic standpoint."

Working along these lines many new and interesting avenues of investigation have been opened by workers the world over. Eckelt[5] found that in those cases exhibiting moderate degrees of intoxication, with albumin, edema and hypertension, there was no nitrogen retention. Losee and Van Slyke[13] showed that in the kidney of eclampsia there was neither marked nitrogen retention, nor severe acidosis, and concluded from this that the toxemia is not a uremia. Zangemeister[16] in his rather exhaustive work on this subject demonstrated that although a definite retention of sodium-chloride occurred in the body during the edemas of pregnancy, the total percentage of all salts excreted in the urine was still relatively high, which proved that no marked insufficiency for salt elimination by the kidney existed. He further showed that in the early stages of the disease there was no functional disturbance in the water output of the renal parenchyma, oliguria occurring only as a later development.

The pathologic picture of a degeneration, the lack of nitrogen retention, the relatively unimpaired salt elimination, the clinical observation that the nephropathies of pregnancy tend toward a spontaneous cure after delivery without causing permanent renal changes, force one to the conclusion that the renal pathology is not responsible for the symptom-complex, that the kidney is not primarily involved, that its function is not seriously impaired until late in the disease, that the changes which occur are of a secondary nature, and that we must look elsewhere for a causative factor rather than cling to the older ideas which are no longer tenable.

The development of the Micro-capillary Tonometer by Danzer and Hooker[4] has given a new impetus to the study of extrarenal factors in all pathologic kidney conditions, especially those connected with pregnancy. It is now possible directly to observe over long periods of time the individual capillary and its tiny blood stream, and to accurately check any deviation from the normal which may exist. Using modifications of this technic, various observers have brought to light some interesting and instructive observations. Working with the capillaries under the finger nail, they showed distinctive changes in both the vessel wall and its fluid contents, in the various degrees of intoxication, found associated with the kidney of pregnancy.

Hinselmann,[9] investigating those cases showing evidence of hypertension, was able to demonstrate a distinct spasm in segments of the capillary wall with a resulting dilatation in other portions of the tube. He was able to observe the contents of the capillaries stagnate with each contraction, the color of the blood gradually changing from red to blue, and in the worst cases all flow ceasing. When relaxation occurred and the flow became reestablished or strengthened the blood again changed color, this time the blue giving way to a normal red, and the overdistention becoming less marked. He found these spasms to be intermittent in character, and their duration and frequency bore a direct relation to the severity of the symptoms. In one very marked case there was complete stagnation for 65 per cent of the time the patient was under observation. An interesting point to be noted is that following delivery the spasms became fewer and of shorter duration, that the overdistended portion of the capillary gradually returned to its normal caliber and that coincident with these changes, the blood pressure began slowly to fall. The same result was obtained, but to a

lesser degree, following appropriate treatment, which will be discussed later. Hinselmann concluded from these findings that the capillary spasm found in the skin was only a part of a generalized vessel contraction throughout the body, and thus gives some support to those investigators who have long held that the kidney changes found in pregnancy are due to a spasm of the blood vessels in the glomeruli.

Nevermann[14] comparing the capillary action in the pregnant and nonpregnant state found that no difference existed, normal gestation evidently causing no change in this portion of the vascular system. As soon, however, as any symptom of an intoxication entered the clinical picture, the findings became abnormal with a resulting stagnation of the capillary blood and a dilatation of the vessel wall. He further showed that venesection with the removal of 300 to 500 c.c. of blood improved the capillary circulation, decreased the number and duration of the spasms, produced a better interchange of blood, and symptomatically seemed to be of benefit to the patient. All of this occurred, however, with a fall in blood pressure of only 20 or 30 mm. of mercury. This observation fits in nicely with the work done by Krogh,[12] who found that capillary tension did not depend on blood pressure, but on the tonus of the capillary wall, and that if stasis existed in the capillary the tonus suffered.

At this time it seems fairly well established that a rise in blood pressure is due to a diminution in the caliber of certain portions of the vascular system, the result of a contraction or spasm of the vessel walls. The causative factor of this spasm is still clothed in utter darkness, which up to the present has defied all attempts at clarification. Some consider it as a part of a general toxic condition associated with pregnancy, but Bumm has shown that one may inject as much as 1000 c.c. of blood from a toxic patient into a nontoxic one and cause not the slightest disturbance. Gessner emphatically denies the existence of a toxic substance circulating in the blood, and points out that any poison which acts over a period of weeks or months must cause changes in the interstitial tissue as well as in the blood vessels. Such changes are not found in the nephropathies of pregnancy, the pathology here being essentially that of a nutritive disturbance of the secreting cells.

The question of edema in pregnancy has from time immemorial been much associated with changes in the kidney substance and only in recent years have we got away from the old dictum, and advanced into a newer and broader understanding of this complicated condition. Zangemeister[16] considers the primary cause of edema to be due to some extrarenal factor which produces a change in the lining of the capillary wall with a resulting transudation of fluid into the tissue. This conception is held by many other investigators, and perhaps typifies the most prevalent attitude of present-day observers. Fink,[6] however, in his rather interesting observations on the causation of edema, sharply attacks this theory, and suggests some very plausible arguments based on the newer conception of the nature of colloids and the laws of osmosis. High blood pressure artificially produced, he maintains, has never caused an edema to manifest itself, even if the vessels were greatly overdistended with fluid, and points out that the interchange of liquid from vessel to tissue is not to be looked upon as merely a filtration process. Edema is not essentially the result of a

kidney involvement, but in this conception depends rather on the quantitative relationship of the organic and inorganic substances in the body to the physiologic processes of the tissue cell. If this relationship is disturbed, molecules and electrones of various substances, ingested or produced by metabolic changes, are set free in the body in excess of cell requirement, and become temporarily stored in various organs, especially the subcutaneous tissue. If the individual cell groups are unable to dispose of these products by speedy elimination they become fixed in the tissue and thus the body colloids are placed in a state where water absorption becomes imperative. We have, therefore, in pregnancy, edemas becoming manifest as soon as a disturbed relationship exists between the molecules presented to the organism by the metabolic processes of both mother and fetus, and the ability of the maternal cells to properly rid themselves by elimination of these excess substances. Fink[6] is in agreement with those authorities who state that edema is responsible for the kidney changes in pregnancy, but differs with them in his conception of the *modus operandi* by which the pathology is produced. He contends that the process is mechanical rather than toxic, that the colloids of the renal parenchyma absorb fluid because of the excess of unused molecules stored in the cell, that the resulting swelling causes pressure to be exerted on the capillary blood supply with interference in the circulation and consequent cell damage, producing albuminuria and cast formation. He considers edema not as a water retention following kidney changes, but water retention with kidney changes, the causative factor of which lies outside the urinary apparatus.

From the brief summary of the literature here presented it will be apparent that the kidney is no longer looked upon as the chief factor in the production of those symptoms which we have come to recognize as part of the picture of the kidney of pregnancy. Most investigators are agreed that the actual changes in the renal parenchyma are secondary, depending upon some extrarenal factor, the nature of which has not as yet been definitely established. The first essential, therefore, in outlining a rational therapy for this class of cases, is an accurate diagnosis, which takes into consideration the possibility of a primary kidney involvement aggravated by pregnancy. Where this unfortunate complication can be ruled out, the treatment as outlined in the following paragraphs would seem indicated.

All authorities are agreed that rest in bed is the treatment *par excellence* for the nephropathies of pregnancy, for by this simple measure the extra work, incident to the erect posture, is taken from the capillaries; the heat regulation becomes simpler, a more even body temperature resulting; less muscular and nervous energy is expended, and the quantity of food and liquid necessary to maintain proper nutrition can be much reduced.

The diet should be bland, avoiding those substances which tend to produce a rise in blood pressure, as spices, coffee and alcohol. Protein and fat can be reduced to a total intake of 60 or 70 grams per day, as the experiences of the Central European countries during the war, when these substances were not easy to procure, demonstrated the advantage of these low figures, in the decrease in the number of severe intoxications recorded. Salt must be restricted to a minimum in any diet prescribed for patients suffering from this condition, as we know

that sodium chloride is retained in the tissues and predisposes to the formation of edema. Liquid intake must be cut down to such a quantity that the body is not overburdened by an excess of fluid which cannot be properly eliminated by the kidneys. A good working rule which may be of service in properly adjusting the amount of liquid ingested is to give only as much fluid per mouth in 24 hours, as the amount of urine excreted in the same time.

From what has just been said it will be apparent that a milk diet, as formerly prescribed, is not suitable for these cases, as milk contains too much water, increasing the fluid intake above the desired amount, and too much salt and protein to come within the required limits. We must rely on carbohydrates to supply the deficiencies in the reduced values of other foodstuffs, and keep up the body weight by a generous supply of such substances as rice, flour, potatoes and sugar, etc.

In those cases which show no improvement under the treatment outlined or where preeclamptic symptoms begin to manifest themselves, special measures must be adopted, the most important of which is venesection. We have seen that the mere taking away of a relatively small quantity of blood is of immense benefit in reestablishing the capillary flow and reducing the angiospasms which are so prominent a factor in this disease. It is, therefore, advisable to take from the circulation 300 or 500 c.c. of blood whenever the indication arises, and to repeat this procedure at short intervals until the desired result is obtained.

The question of the termination of pregnancy is one which hardly comes within the scope of this review, inasmuch as we have limited ourselves to the consideration of the kidney of pregnancy *per se* without taking up either eclampsia or true nephritis. It is selfevident that where no improvement can be brought about after a prolonged trial of the above mentioned measures, and where with the most careful attention the patient rapidly becomes worse, the uterus must be emptied.

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Book Reviews

The Place of Version in Obstetrics. By Irving Potter, M.D., F.A.C.S. C. V. Mosby Co., St. Louis, 1922.

This monograph containing a foreword and seven chapters, deals successively with the History of Version, the Present-day Employment of Version, the Author's Technic of Version, besides some twenty-three pages devoted to Criticisms and Answers, Indications and Advantages, and statistical reports for the years ending August 31, 1920, and 1921, with conclusions drawn therefrom.

In the foreword Dr. Potter defines his position in reference to the performance of version, when he states, that, "from an experience extending over many years of practice, including thousands of personally conducted cases of labor, I have come to believe that it is justifiable to perform a podalic version for the purpose of eliminating the second stage of labor and relieving the woman of the pains and agonies of childbirth, since such a procedure in my hands has been attended with no increase of fetal mortality and has had a lessened maternal mortality and future morbidity."

After reading this little book, one cannot help being impressed with the fact that the practice of obstetrics in Potter's hands divides itself into version and cesarean section, interrupted by an occasional spontaneous delivery occurring before the arrival of the attendant. That this is radicalism does not admit of argument.

One is further impressed with the fact that the women of Buffalo are physically and psychically different from those found in other parts of the country, in that they apparently do not suffer pain during the first stage of labor. For the parturient elsewhere, the stage of dilatation is often the most prolonged, painful and tedious part of the labor, the one that exhausts and is not forgotten, for the second stage in modern practice can be and is made relatively painless by the use of analgesia and anesthesia. Hence the principal indication for version as set forth by the author, i.e., to eliminate the pain of the second stage of labor, fails to relieve the woman of the agonies of childbirth.

The forty odd pages describing the author's technic in version are well written and illustrated with many explanatory photographs and drawings. This chapter should be carefully read and the details contained therein applied in the practice of every obstetric surgeon. The importance of skilled anesthesia to secure proper muscular relaxation and the employment of the modified Walcher position are practical points of no little significance.

Furthermore, credit must be given to Potter for what he has done for obstetrics in perfecting the technic of internal version and extraction, for in this book he has presented to us a logical succession of steps, based on the fundamentals, that is an improvement on all former procedures.

After reading every sentence of this monograph we are as yet in ignorance as to why version was performed 920 times in 1113 deliveries in 1920 and 938 times in 1130 deliveries during 1921, unless it was for the convenience of the operator or on that questionable indication, eliminating the pain of the second stage of labor. It must be remembered that an operator of Potter's skill, experience, manual dexterity, and indefatigable energy, who has perfected himself in the details of one

procedure must necessarily look upon the wider indications for version with bias and his results cannot be justly comparable with those of others with fewer opportunities; hence his indications cannot be admitted even on his statistics.

As a whole the book is a valuable contribution on the subject of version and the chapter on technic deserves the consideration of every obstetrician.

JOHN OSBORN POLAK.